AUG 17 1944

Medical Library

BRITISH MEDICAL JOURNAL

JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

SATURDAY JULY 29 1944

TABLE OF CONTENTS

Localization in Cerebrum and Cerebellum E. D. ADRIAN, M.D., F.R.S. 137

Problems in Riboflavin and Allied Deficiencies—II HUGH S. STANNUS, M.D. 140

Post-operational Strain in the Navy DESMOND CURRAN, M.B., and GERALD GARMANY, M.B. ..144

Schick Reactions in Recently Confined Women and their Infants G. PAYLING WRIGHT, D.M., and W. M. CLARK 146

Radiotherapy of Ectopic Calcification E. MILLINGTON, M.R.C.S. 148

Some Common Paediatric Procedures DOYNE BELL, D.M. 157

MEDICAL MEMORANDA

General Hypersensitivity to Sulphanilamide after Surface Application. G. G. WALLIS, M.B. 149

Weil's Disease without Jaundice. ALICE STEWART, D.M., and L. J. WITTS, D.M. 149

A Case of Fat Embolism. G. ST. J. HALLETT, F.R.C.S., and A. J. M. DRENNAN, M.B. 150

Torsion of the Gall-bladder. E. WILSON HALL, F.R.C.S.Ed. 150

GENERAL ARTICLES AND NEWS

Lister Institute Report 158

Reduction of Leg Fractures by Hydraulic Tibia Traction. J. E. STANLEY LEE, F.R.C.S.Ed. 152

ORDER OF ST. JOHN: Promotions and Appointments 159

THE SERVICES 164

UNIVERSITIES AND COLLEGES 165

MEDICAL NOTES IN PARLIAMENT 165

MEDICAL NEWS 166

LETTERS AND ANSWERS

Haemorrhage after Gastro-jejunostomy—Tenderness after Shaving—Liver Stores and Ketosis—Hallucinations and Heart Failure—Treatment of Anxiety Symptoms—Galactorrhoea—Aniline Poisoning—Tap Water and Blood Transfusion—Sunlight and Tuberculosis—Cysts of Buccal Mucous Glands—Income Tax—Sterilization of Syringes—Baby wanted for Adoption—Hospitality for Doctors' Children—Flying Bomb: Exchange of Duties 167-8

LEADING ARTICLES

Problems of Ariboflavinosis 153

The "Triple" and the "Conjoint" ...154

ANNOTATIONS

Surgical Treatment of Coronary Disease 155

Health Record of U.S. Army 156

Medical Films 156

REVIEWS

Record of Medical Progress 151

Rheumatic Fever and Chronic Arthritis 151

Parodontal Disease 151

Notes on Books 151

OBITUARY

Sir Hugh Rigby, BT., K.C.V.O., F.R.C.S. 163

Sir William Lister, F.R.C.S. 163

R. N. Wilcox, M.B., D.L.O. 164

Ross Millar, M.D., C.M. 164

F. W. Hope Robson, M.D. 164

Frederick Henry, M.B. 164

SUPPLEMENT

"Public Relations": The Profession and the Press 21

THE QUESTIONARY 21

CORRESPONDENCE 23

BRANCH AND DIVISION MEETINGS 24

H.M. FORCES APPOINTMENTS 24

POSTGRADUATE NEWS 24

CORRESPONDENCE

B.M.R. Determinations on Ambulatory Patients. I. M. RABINOWITCH, M.D. 159

Why Tie the Cord? IAN W. MONIE, M.B. 159

Perineal Tears in Normal Confinements. H. E. HUMPHRYS, M.B. 160

Infant Feeding. P. R. BOUCHER, M.B. 160

Milk for School-children. J. ALISON GLOVER, M.D. 160

Estimation of Dental Caries. GUY NEELY, M.R.C.S., L.D.S. 160

Stomatology. C. BOWDLER HENRY, M.R.C.S., L.D.S. 160

Pentothal Anaesthesia. F. W. ROBERTS, M.B. 161

Sodium Pentothal in Nasal Operations. E. R. DINGLE, M.B. 161

The Cold Agglutinins. F. STRATTON, M.D. 161

Intravenous Barbiturates in Hysteria. F. DILLON, M.D. 161

Chemotherapy in Otitis Media. C. CARRON BROWN, M.D. 162

Mastoiditis and Mastoidectomy. H. HILTON BROWN, M.B. 162

Sulphonamide Prophylaxis of Gonorrhoea. G. L. ALEXANDER, M.B. 162

Gonorrhoea in North Africa. J. F. PEART, F.R.C.S.I. 163

Stilboestrol in Cancer. J. T. SHIRLAW, M.D. 163

Tarso-metatarsal Dislocation. D. A. RICHMOND, F.R.C.S. 163

Epidemiology Section 166

BRITISH MEDICAL ASSOCIATION, TAVISTOCK SQUARE, LONDON, W.C.

No. 4360, WEEKLY, PRICE 1/6

Copyright

REGISTERED AS A NEWSPAPER

Respiratory Emergencies!

SPARKLET RESUSCITATOR

The 'Sparklet' Resuscitator has again and again proved invaluable in the Carbon Dioxide treatment of Respiratory Failure Emergencies.

CO_2 Snow Sticks!

POCKET CO_2 SNOW OUTFIT

With this outfit, CO_2 Snow Sticks are prepared in a few moments, and the treatment of skin blemishes made convenient and economical.

Write for special booklets to Dept. 43

SPARKLETS LIMITED
LONDON, N.18.

Present-day food policy

- The guiding principle of everybody nowadays should be to make the best possible use of available foodstuffs from the nutritional stand point.
- Yeast has long been appreciated for its food value and its usefulness as a source of the B vitamins. It is for this reason that Marmite—an autolysed extract of fresh brewers' yeast—is being ordered increasingly for its nutritive properties.

MARMITE
YEAST EXTRACT

The Marmite Food Extract Co., Ltd., 35, Seething Lane London, E.C.3

446

THE NERVOUS HEADACHE

Present-day conditions stress the need for an analgesic which may be taken with the minimum of habit induction or other after effects.

'Anadin' may be administered with safety over an indefinite period where the depressing action of the barbiturates and other potent sedatives would result in lowered efficiency and ultimately in impairment of the excretory mechanism.

Dose:
Two tablets at four hourly intervals.

Formula: Acetphenetidin 3 gr.; Ac. Acetylsalicylic. 4 gr.; Caffeine Alkaloid. ½ gr.

ANADIN ***Tablets***

ANADIN LIMITED, 12, CHENIES STREET, LONDON, W.C.1

'AVLON'
brand of
PETHIDINE HYDROCHLORIDE

Pethidine Hydrochloride is indicated in the treatment of neuritis and spastic conditions of the renal and biliary tract. Pethidine is also of value where prolonged labour is due to cervical spasm, but is contra-indicated in the presence of toxæmia or hypertension.

Further information will be forwarded on request

PACKINGS

Tablets of 0.025 *gramme and* 0.05 *gramme in bottles of* 25, 100 *and* 500.
Ampoules of 0.05 *gramme* (1 *c.c.*) *in boxes of* 12 *and* 100 *and* 0.1 *gramme* (2 *c.c.*) *in boxes of* 10 *and* 100.
Bottles of 25 *c.c., each* 1 *c.c. containing* 0.05 *gramme.*

Obtainable from your usual suppliers.

A Product of
IMPERIAL CHEMICAL [PHARMACEUTICALS] LTD.
Commercial Department: 89, Oxford Street, Manchester, 1

Ph 15g

BRITISH MEDICAL JOURNAL

LONDON SATURDAY JULY 29 1944

LOCALIZATION IN THE CEREBRUM AND CEREBELLUM

BERTRAM LOUIS ABRAHAMS LECTURE*

BY

E. D. ADRIAN, O.M., M.D., F.R.S., F.R.C.P.

Professor of Physiology, University of Cambridge

Bertram Louis Abrahams, in whose memory this lecture was founded, was a medical student in the early 'nineties. He was a Londoner born and bred, had a brilliant career with a University scholarship in physiology and later a registrarship at the Westminster Hospital, and ended as lecturer in physiology and as assistant physician there. He was only 38 when he died, but he had an established reputation as a consultant and a teacher, and he had lived a life full of intense interests and activities—and not only in the field of medicine, for he was an ardent cricketer and Freemason and the organizer of a boys' club. But one of his chief interests was physiology, and so it is fitting that his name should be associated with a lecture on a physiological theme.

One way in which we might call him to mind would be to discuss and so to re-create in ourselves some of the thoughts and ideals which seem to have guided his work. Since he was a teacher of physiology as well as a clinician, he must often have had to face the perennial question of how physiology should be taught as a basis for medical work. If we dealt with this we might think of ourselves as carrying on, as it were, a discussion in which Abrahams must often have taken part 40 years ago. But I am afraid it would be found that there was little new to add to the discussion in spite of all the good advice which has been given from all sides in the past 40 years. The details of the picture have changed, but in its outlines the problem will remain as long as physiology is taught.

Another theme in which Abrahams was keenly interested was that of the general physiological principles which govern the reactions of the cell or the organism to injury. But here the picture has changed too much. Nowadays the physiologist has few principles left; for the emphasis has passed from the general to the particular, from the katabolism of protoplasm to the details of carbohydrate cycles. Moreover, in these uncertain times I think most of us will prefer facts to theories, however narrow the field from which they have come: so I have chosen a very limited branch of physiology, of which the most important thing we can say is that it is going ahead.

The Clinician and Cerebral Localization

The main facts of cerebral localization were established during Abrahams's lifetime and were as much the achievement of clinical medicine as of physiology. Neurology has always encouraged a very close interchange between the clinical and the laboratory worker: in this country, for instance, we have had clinicians like Hughlings Jackson, physiologists like Schäfer and Sherrington, and men who were both, like Ferrier and Horsley. There are obvious reasons why both kinds of investigation are needed for the brain. It is the organ in which there is the greatest difference between man and other mammals, so that the findings from animal experiments do not necessarily apply to man and must be checked and modified by observations on patients; and men can give us far more information than animals in that we can communicate with them and find out what they feel and think. On the other hand, there are many problems involving the motor area in which results can be obtained with far greater certainty from animals than from human disease or accident.

The result of this was that until recently we knew more about the motor area in the monkey and more about the sensory and visual areas in man, though it had taken the head wounds of the last war to provide all the necessary clinical material. Since that war, however, there have been two important developments. One is the outstanding progress of neurosurgery, which has made it a much less formidable business to expose the human brain and has given us maps of the motor cortex in man to set beside those of the chimpanzee. The other development is in the use of electrical methods to investigate the brain in animals, and this has given maps of the receiving areas to compare with the clinical findings in man. I shall describe some of the information obtained by this method, for it is a relatively new physiological technique which is only at the beginning of its usefulness.

A New Physiological Technique

The technique has been made possible by the use of better electrical recording instruments and more suitable anaesthetics. The former depend on valve amplification and need not be considered in detail; the anaesthetics are the barbiturates. These have an action on the brain quite unlike that of ether or chloroform, for they reduce the persistent activity of the cortex (shown by persistent electrical waves) without preventing the arrival of the messages from the sense organs. These messages produce their own electrical accompaniment, which can be recognized without difficulty provided that it occurs against a quiet background. Thus the barbiturates allow us to record what comes into the afferent areas of the brain with a precision quite impossible with other anaesthetics which have a less selective action. In fact, their main disadvantage is that we tend to rely too much on them and to forget what they may be doing to the brain by suppressing all but the simplest activities.

What can be done with this kind of technique may be illustrated by the mapping of the visual area of the monkey's brain by Talbot and Marshall (1941). A thin pencil of light is directed to a particular spot on the retina while the striate area of the cortex is explored with electrodes leading to an amplifier and oscillograph. It is found that turning the light on and off produces electrical activity in a very small part of the striate area and that any change in the position of the illuminated spot on the retina is reflected in a corresponding change in the region of electrical activity in the brain. There is, in fact, a precise correspondence between retinal and cortical points which allows us to make a definite map of the retina as it is projected

* Delivered to the Royal College of Physicians on June 8, 1944.

on to the surface of the brain (Fig. 1). Points near the centre of the fovea are connected with the antero-lateral margin of the striate area, and points nearer the periphery with the posterior, mesial part. Thus, allowing for the fact that in the human brain the whole striate area has been pushed back to the mesial aspect of the hemisphere, the connexions between the retina and striate area in the monkey agree with the connexions established for man by Holmes and Lister from the gunshot wounds of the last war.

It is satisfactory to have the human findings confirmed by such a different sort of evidence, and, although the result was not at all unexpected, the important point is that the mapping can be done with a very high degree of precision, so that conclusions can be drawn as to the mechanism of visual discrimination and the like. In the anaesthetized brain, therefore, we can say that a pattern of light and shade thrown on the retina will produce a corresponding pattern of activity in the afferent fibres which enter the striate area. What would happen in the area if there were no anaesthetic is another matter. Even in the simplified conditions given by deep anaesthesia, where all but the most direct pathways are out of action, there is a good deal of editing of the messages on the way up to the brain, and if there were no anaesthetic the pattern arriving there might be much less recognizable because of interactions between neurones at the various synaptic levels on the visual pathway. And the fact that the pattern in the striate area may be recognizable to the physiologist with his electrical equipment is of little help in explaining how it is recognized by the brain in which it appears.

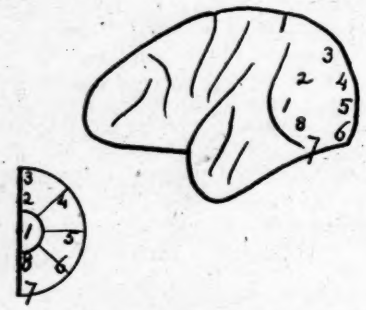


FIG. 1.—Representation of the retina in the visual area of the monkey's brain.

Nervous Mechanism of the Cochlea

It would have been surprising if a spatial correspondence between retina and striate area had not been found—but until recently there was less reason to expect such a thing in the auditory area, and here the electrical method has been particularly illuminating. Within the past two years the nervous mechanism of the receptor organ, the cochlea, has become far clearer, so that we have a much better notion of the sort of messages which it transmits to the brain. They have been studied by Galambos and Davis (1943) from records made from single fibres of the auditory nerve. It was found that each nerve fibre comes from receptors tuned to a particular pitch but capable of reacting to louder notes if their pitch is not far different. This agrees with the familiar Helmholtz conception, but Galambos and Davis have added the interesting point that the message which the auditory nerve fibre transmits to the brain is not itself marked by any particular frequency. It is simply a train of impulses whose frequency varies with the intensity of the stimulus; in fact, it is like all the other sensory messages which reach the brain, and could not be distinguished from them if we did not know that it came from the cochlea and so must signal sound rather than sight or touch.

Now if the pitch of the note can only be inferred from the fact that it has excited the receptors in a certain part of the basilar membrane, it follows that there must be some kind of spatial representation of the cochlea in the brain just as there is of the retina. And it has been found, first in the cat and now in the monkey, that such a representation does exist. When the cochlea is stimulated by sound, electrical activity appears in the appropriate region—the upper surface of the superior temporal gyrus—and with notes of low pitch the maximum activity (in the monkey) is in the antero-lateral, with high notes in the postero-median, part of the receiving area (Licklider and Kryter, 1942). Thus the apex of the cochlea supplies the antero-lateral, and the basal turn the postero-median part (Fig. 2). Just as the movement of a light to and fro across the visual field will produce a peak of activity moving to and fro across the striate area, so the up-and-down note of

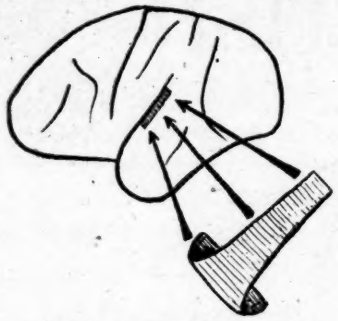


FIG. 2.—Representation of the basilar membrane of the cochlea in the auditory area of the monkey's brain.

the siren will give an activity moving to and fro in the cortex of Heschl's gyrus. To have reached this position is a distinct achievement, and it has cleared the ground for the attack on the main position—the problem of why a nervous signal passing to and fro in the auditory cortex with particular time and intensity relations should start in us the whole train of thoughts and actions appropriate to air raids. But here the attack has scarcely begun, and the position is defended in very great strength.

Relation of Brain to Sense Organs

The signals which reach the brain from the peripheral sense organs arrive in the post-central gyrus (in man and monkey) in the positions we should expect; messages from the hind-limb receptors appearing near the vertex, those from the fore-limb further down laterally, and so on. Again we have a strip of cortex with peaks of activity here and there corresponding to the stimulated sense organs, and we infer what is happening to our bodies from the rise and decline of the messages in different parts of the area. But as the electrical method allows us to deal with animals of all sorts it has brought out some interesting differences in sensory equipment, or rather in the importance to the brain of the sense organs from different parts. For instance, if we compare the receiving areas in the brain of the cat and the pig we find that in the cat there is a large fore-limb area with exact representation of the digits, foot-pad, etc., whereas in the pig the area chiefly represented is the snout, and it is difficult or impossible to detect anything at all from the limbs (Adrian, 1943). The reason is presumably that the cat uses its fore-limbs and claws in skilled movements which

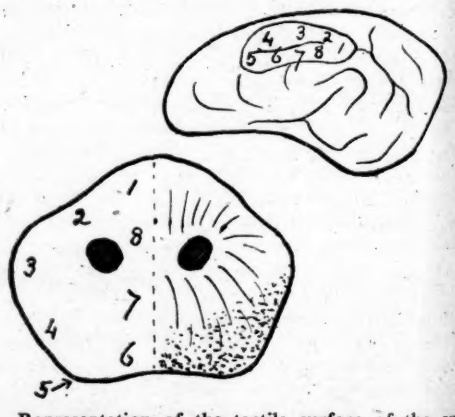


FIG. 3.—Representation of the tactile surface of the snout in the sensory area of the pig's brain.

must be precisely guided by sight and touch, whereas the pig uses its limbs for standing and walking, and for nothing else. All the skilled movements of the pig are made by the snout, and the snout has an elaborate representation in the brain with a precise correlation between points on the snout surface and points in the cortical receiving area (Fig. 3). In fact, judging by the size of the cortical area in relation to the skin surface

supplying it, the pig's snout is represented on a scale even more generous than that of the human hand. Thus the snout takes a place as one of the special receptive surfaces like the retina and the basilar membrane.

In other hoofed animals there is the same preponderance of afferent signals from the snout region and neglect of those from the limbs; but no one can use this method of examining the brain without realizing the importance of the sense organs in this region in every animal but man. It is implied in Sherrington's conception of the leading segments, and is shown not only by the area devoted to it in the brain but by the size of the fifth nerve and the special types of receptor organ supplied by it. The tactile vibrissa of the snout is a good example of an organ which might really be classed as a distance receptor, for one has only to look at the head of a rat to see how the snout, by the aid of its long vibrissae, comes to be literally in touch with objects more than the width of the head away. In man the sensory mechanism of the fifth nerve has lost this function of exploring space, but there are still the large number of sensory fibres to the face and its acute sensitivity to touch to remind us of what is gained and lost by the erect attitude.

The Role of the Cerebellum

These are only a few of the points which have been established in regard to the cerebrum, but it is time to pass to a different field. Few neurologists of my generation can have forgotten the satisfaction induced by Gordon Holmes's analysis of the symptoms of cerebellar injury. It was almost the only sign that that mysterious organ the cerebellum had any intelligible function at all. But until quite recently the clinical findings have remained more illuminating than anything from anatomical or experimental sources, and the cerebellum has kept its position as one of the most puzzling parts of the nervous system. The newer methods have now brought distinct signs of improvement; for we have at last begun to get fairly good evidence of localization of function, or at any rate localization of nervous mechanism, in the cerebellar cortex.

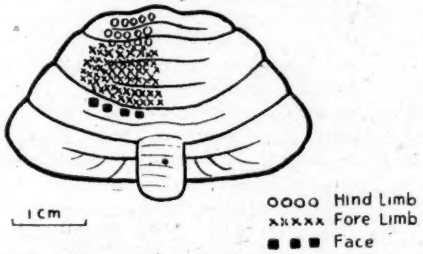


FIG. 4.—Receiving areas for spino-cerebellar impulses in the cerebellum of the monkey.

A scheme of cerebellar localization was put forward some time ago by Bolk on morphological grounds. It made the anterior lobe responsible for the head, and the ansiform lobes for the arms; but since then anatomical evidence has shown that the afferent supply from the vestibular nucleus goes mainly to the posterior region and that from the spinal cord to the anterior. This in itself indicates some differentiation of function, and, by stimulating the various tracts electrically, Dow, who is largely responsible for the recent interest in the cerebellum, has verified the general distribution of the different sets of afferent fibres.

But a good deal more can be made out when we explore the cerebellar cortex with a fine needle electrode so as to pick up the impulses as they arrive in the white matter. To obtain a clear picture either the spino-cerebellar or the ponto-cerebellar tracts must be put out of action, since their distribution overlaps to some extent; but when one or the other is suppressed an arrangement is revealed which is strikingly reminiscent of that in the sensory or motor area of the cerebrum.

The arrangement in the monkey is shown in Fig. 4. The marked regions show where messages arrive in the anterior lobe from the spino-cerebellar tracts when the limbs on the same side of the body are pressed or moved. The map is quite different from that of Bolk; for messages from the hind-limb go furthest forward, those from the fore-limb go to the culmen. Messages from the face appear in the lobulus simplex, but are not always found, and when they are they come not from neck muscles but from the vibrissae of the face—possibly because the vibrissae have a special function in guiding the head.

The messages to the anterior lobe from the limbs are partly tactile, but most of them come from the deeper endings in muscle and tendon and in the fascia of the hand and foot. The most effective stimulus is always a pressure on the pad of the foot combined with dorsiflexion of the digits—as though the limb were to take the weight of the body—but all kinds of joint movement and stretching of muscles have been effective. The localization is like that in the cerebrum, with the arrival areas arranged in the order from behind forward for hand, wrist, elbow, shoulder, hip, knee, ankle, and foot.

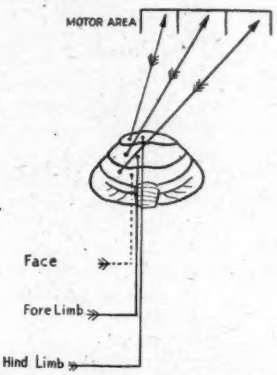


FIG. 5.—Spino-cerebellar and ponto-cerebellar pathways to receiving areas in the anterior lobe and lobulus simplex.

It is not possible to map out the distribution of the ponto-cerebellar fibres in such fine detail, but by stimulating various points on the motor cortex and picking up the discharges which are relayed to the cerebellum through the pons we can find an arrangement which is substantially the same, the hind-limb area of the motor cortex sending impulses to the most forward part of the cerebellar area, etc.

The ponto-cerebellar areas extend further out laterally than the spino-cerebellar, and there is a large region further back which does not seem to correspond to any one part of the cerebral cortex, but has a diffuse connexion with scattered points in it. For the present the relation of this relatively large ponto-cerebellar receiving area to the cerebrum must remain uncertain, and all that can be said is that there are these localized receiving areas in the anterior lobe for the different segments of the body arranged as in the diagram (Fig. 5). Probably there are other arrival and departure areas as well; indeed, in the rat (Dow and Anderson, 1942) impulses from the limbs arrive in the pyramis further back, and some of the spino-cerebellar fibres end there in the cat and monkey. But although we may be dealing with only one side of the picture it is worth illustrating it in another way—namely, by comparing the size of the culmen (i.e., the part of the anterior lobe concerned with fore-limb messages) in three different animals—man, the pony, and the pig (Fig. 6). The comparison is more than usually speculative because the evidence on which it is based is mainly derived from the cat and monkey, but the three animals are chosen because they do not differ

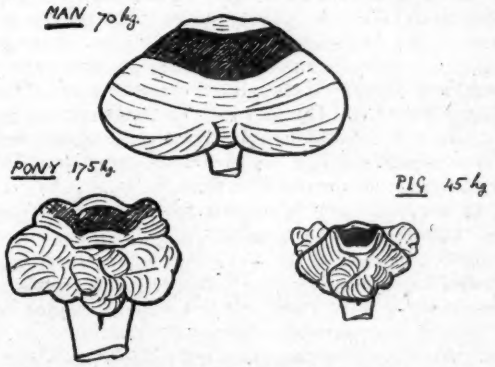


FIG. 6.—Comparison of the culmen (receiving area for the fore-limb) in man, the pig, and the Shetland pony.

very greatly in size and weight but do differ in the use to which the fore-limb is put. There is evidently a great difference in the extent of the cerebellar area to which impulses concerned with the fore-limb are sent; and the comparison is also useful because it draws attention to the large fraction of the cerebellum which does not seem to be employed in any localized representation of the body and limbs. It shows, in fact, that we are still at the beginning of the story.

Conclusion

I have tried to give a sketch of a method of research on the central nervous system in which rapid advance is possible and in which it is difficult to foresee how far it may not go. But, as with all long-range research nowadays, the advance has been held up until the younger generation of physiologists can come back from the more urgent problems which they have to solve. Some day military or naval or air physiology should form the subject of a future Abrahams lecture. Compared with these problems of life and death the academic study of the nervous system seems to-day of very little consequence. All I can hope is that an account of its progress may serve as a reminder of the things which were important to us in better times, and as a reminder of a Fellow of the College of Physicians who lived when those times were taken for granted as the natural consequence of civilization.

Figs. 4 and 5 are reproduced by kind permission of the Editor of *Brain*.

REFERENCES

Adrian, E. D. (1943). *Brain*, **66**, 89.
Dow, R. S., and Anderson, R. (1942). *J. Neurophysiol.*, **5**, 363.
Galambos, R., and Davis, H. (1943). Ibid., **6**, 39.
Licklider, J. C. R., and Kryter, K. D. (1942). *Fed. Proc. Amer. Soc. exp. Biol.*, **1**, 51.
Talbot, S. A., and Marshall, W. H. (1941). *Amer. J. Ophthal.*, **24**, 1255.

SOME PROBLEMS IN RIBOFLAVIN AND ALLIED DEFICIENCIES*

BY

HUGH S. STANNUS, M.D., Ph.D., F.R.C.P.

(*Concluded from page 105*)

SIGNS AND SYMPTOMS OF HYPORIBOFLAVINOSIS

The mild constitutional symptoms, including the feeling of being "out of sorts," of loss of "pep," of mental and physical fatigue, might thus be explained, as also perhaps symptoms referred to the heart and gastro-intestinal tract.

All the dermal manifestations support my thesis, I believe. The skin has been regarded as a tissue possessing but a relatively small capillary vascularization, associated with a low metabolism, but this is fallacious. The first statement referred to the number of capillary loops per square millimetre which can be counted by Lombard's method of microscopy and left out of consideration the subpapillary plexus—the plexus which gives to the skin its colour and which itself is made up of giant capillaries. The second statement was based on the tissue-slice respiratory method of estimation, when of course the skin was not exercising any of its normally marked activities. The skin as a whole may be affected, or only those areas whose anatomico-physiological design is specialized in some direction, as I pointed out some years ago.

A mild erythema associated with fine desquamation, referred to by Sydenstricker as pityriasis sicca, may be remarked. It is most easily recognized in the skin of the forehead and face, sometimes on the hands, in the fair-skinned individual. In the pigmented skin of the native the erythema cannot be detected, but the desquamation may be noted.

In other cases the change may be obvious only where the skin is very *thin*, as in the antecubital fossa, or *specialized*, as in the case of the scrotum and in the transitional type of skin about the orifices of the body—the palpebral fissures, the nares, the free margin of the prepuce, the vulva and anus—giving rise to the lesions I have described.

Sydenstricker two or three years after his first communication on ariboflavinosis mentioned pruritus of the scrotum and vulva, but made no reference to any visible lesion. On the other hand, an excellent photograph of the scrotal condition was reproduced by Spies and Cooper (1937), but with the subscription "pellagrous dermatitis of the scrotum and thighs." The lesions of the prepuce, vulva, and anus do not appear to have been recognized by American observers. Possibly they have not distinguished them from the urethritis, vaginitis, and proctitis which occur in pellagra.

The seborrhoeic lesions about the nose and in the naso-labial folds are a good example of the disturbance in an area of skin which normally possesses a rather different anatomico-physiological arrangement from the rest of the skin. Whether these lesions are of the same nature as those described by Smith, Smith, and Callaway (1941) under the designation "dyssebacia" is uncertain, as they held that the condition they described was not cured by any one of the known vitamin B complex factors but did respond to autoclaved yeast or liver extract.

The affection of the vermilion portion of the lip—cheilosis—is another example of the incidence of the lesion being associated with anatomical specialization, for here the capillary loops are notably very much closer together than in other parts of the skin.

The best description of the changes in the skin capillaries is contained among observations made by Márquez Blasco and Peraita (1940) upon patients suffering from nutritional deficiencies during the Spanish Civil War. They found that, using a dermal capillaroscope, pathological changes, least marked in simple cases of pellagra, were progressively more accentuated in proportion to the advent and advance of symptoms indicating involvement of the nervous system, including loss of visual and auditory acuity—symptoms which I shall come to shortly.

Angular Stomatitis

The angular stomatitis calls for some words of comment. The term—not a very happy one—was originally used by myself, and seems unfortunately to have persisted. It is difficult to find a better as, so far as I know, there are no words in any language which distinguish between the opening into, and the cavity of, the mouth. Angular stomatitis at least serves to distinguish the lesions so named from "cheilosis," the lesion of the vermilion of the lip, though some American authors—wrongly, I believe—include both under cheilosis.

If the corners of the mouth be examined carefully, with the mouth open but produced into a pout, it will be observed that the vermilion portion of the lips does not encircle the mouth at the angles, but passes internal to the delicate fraenum which has become visible—that, in fact, the lips are prolonged on to the inner side of the cheek. This accounts for another common feature in this condition—namely, the presence on the inner side of the cheek of a linear lesion comparable to the angular stomatitis on the skin outside.

That the lesions of riboflavin deficiency become worse if nicotinic acid be given has been noted by several observers without any explanation. I suggest it is due to the pharmacological vasodilator action of nicotinic acid on an already damaged capillary endothelium.

These lesions are, I believe, characteristic, and with experience I think it may be possible to distinguish them from others produced, it has been suggested, by dentures, etc., and from the condition sometimes to be seen in those with turned-down corners to the mouth, patches of herpes, impetigo, lichen, etc. The introduction of the expression "pseudo-ariboflavinosis" by Ellenberg and Pollack (1942) is, I think, much to be deplored.

This leads me to mention the word "perlèche," a term adopted from the French which one would like to see banished from our medical literature. As pointed out a few years ago by myself (1941) in dealing with the history of this affection, it has become a habit—a bad habit, copied by one author from another, in ignorance of its original significance—when referring to angular stomatitis, to give as a synonym this foreign word. It was first used by a French doctor named Lemaistre in 1886 as the title of an article—"*De la perlèche: du streptococcus plicatilis.*". He described a condition—which closely resembles angular stomatitis—that he had observed to be common among children in the neighbourhood of Limoges,

*The Lumleian Lectures (abridged) delivered before the Royal College of Physicians of London, April 18 and 20, 1944.

where it had long been recognized by the inhabitants under the vulgar term "perlèche"—derived from the verb *lécher*, to lick, as it had been noticed that the sufferers had the habit of "feeling for" the sore place with the tip of the tongue. Lemaistre believed the disease was due to a streptococcal infection and that it was contagious, as some 10 to 30% of school-children suffered from the complaint.

Subsequent authors, influenced by its supposedly streptococcal origin, used the word perlèche to denote an impetiginous affection of the corners of the mouth. If, therefore, perlèche were and be an impetiginous affection the word should not be used for a lesion due to hyporiboflavinosis. On the other hand, it seems to me very possible that the Limoges children were suffering from riboflavin deficiency, in which case the condition, defined as due to a streptococcus, was incorrectly described. In either case to retain the word is only to confuse the issue.

It is of interest in this connexion, however, to note that cultures prepared by Riddle, Spies, and Hudson (1940) from lesions at the angles of the mouth in cases of riboflavin deficiency yielded a pure growth of *Staph. aureus* in 80% and *Str. haemolyticus* in 20%, and that the organisms disappeared when the lesions healed with riboflavin therapy.

The angular stomatitis occurring in sprue appears to be due to riboflavin deficiency; some uncertainty still exists as to whether the similar condition seen in simple achlorhydric anaemia or idiopathic hypochromic anaemia and in that type presenting the Plummer-Vinson syndrome is due to the same cause. Some observers (Aykroyd, Smith and Martin, Rosenbaum and Jolliffe, Machella, Spies, etc.) have failed to obtain a satisfactory response to riboflavin therapy. I have met with similar cases, but it must not be forgotten that a considerable increase in dosage or administration by parenteral route may be necessary before an effect is obtained.

In regard to the glossitis, Sydenstricker and his colleagues have said: "The tongue is clean, the papillae flattened and mushroom-shaped and hypertrophic, the colour is definitely purplish red or magenta." This tongue is certainly characteristic, but whether of diagnostic value alone seems doubtful. By microscopy the filiform papillae are seen to have lost their keratinized tip, they are mushroomed out, swollen, and slightly oedematous, forming a pavement of semi-transparent hemispheres like minute jellyfish, while in the centre of each may be seen a dilated and distorted capillary loop, comparable to those mentioned as occurring elsewhere. Later, irregular patchy denudation may occur, giving rise to a condition which has been called "geographical tongue."

Ocular Manifestations

The ocular manifestations of hyporiboflavinosis have received a good deal of attention of recent years. Careful studies of the effects of deficiency states on the eyes of experimental animals began with the work of Wolbach and Howe (1925) upon vitamin A deficiency in the rat and the production of corneal vascularization, a condition resembling interstitial keratitis and cataract.

In 1939 Bessey and Wolbach published their work on vascularization of the cornea in riboflavin-deficient rats. In the same year Sydenstricker and his co-workers, after the original communications by Sebrell and Butler, described the symptomatology of "ariboflavinosis," but no mention was made of ocular signs and symptoms. The following year saw the first reference to eye lesions, and a more elaborate communication appeared on the subject by Sydenstricker and his colleagues. The objective signs described included injection of the bulbar and palpebral conjunctiva and of the fornix, circumcorneal injection, engorgement of the limbic plexus, and vascularization of the cornea; further, superficial opacities and deeper interstitial changes, sometimes diffuse, sometimes patchy, were noted, also congestion of the sclera and iris, and in some cases frank iritis. The authors concluded that "keratitis" is the principal ocular lesion of ariboflavinosis: the earliest and most common sign is circumcorneal injection.

The full realization of these lesions became possible only when slit-lamp microscopy was introduced for their study, a fact which probably explains their non-recognition in the older syndrome, more especially as the subjects were all coloured peoples with very dark irides.

Apart from the symptoms referable to these conditions—"itching and burning sensations," "roughness of the eyes," and photophobia—the one subjective symptom in all these cases was impaired visual acuity, referred to as dimness of vision or partial blindness, associated with mydriasis.

Sydenstricker remarked: "Functional disturbances of vision out of all proportion to visible changes in the cornea were present in a number of patients with oral and lingual as well as ocular lesions of ariboflavinosis," but he offered no explanation. I think American observers have failed to realize the significance of the *subjective* symptoms as distinguished from the *objective* signs. All the congestive signs I look upon as part of the general capillary dysergia, comparable to those in the skin but so situated as to be freely open to examination. On the other hand, the visual disturbances I believe to be due to a lesion in the nervous system with no immediate connexion with changes in the eye. That this should be so appears never to have occurred to any of those who have published their findings; that some condition akin to a retrobulbar neuritis would best explain the symptom has never been considered apparently by American observers. I shall refer to this question later.

There are other observations recorded from the United States and elsewhere which are based, I venture to think, on misconceptions in regard to the normal. In the article to which I have already referred mention is made of a "striking increase in the number of vessels composing the limbic plexus" and of replacement of "the normal narrow avascular zone between the limbic plexus and the sclero-corneal junction by a dense capillary network," etc. Elsewhere the statement is made: "For reasons not apparent arcus senilis would seem to offer some obstacle to the extension of the capillaries beyond the sclero-corneal junction."

This is not the place, perhaps, to discuss ophthalmological minutiae; but inaccuracies have already crept into the literature on the subject, as in H. Jegher's *Advances in Internal Medicine* (1942) and some of our own textbooks, and are in danger of being perpetuated, so that some reference must be made to them.

My own observations—and I speak with considerable experience, having, with my colleague Prof. Brunel Hawes, examined by slit-lamp microscopy the eyes of between four and five thousand individuals, aged from 3 to 93 years of age, healthy and sick, with and without ocular disease—lead me to believe that there is always a complete limbic plexus and there is no narrow avascular zone between the limbic plexus and the sclero-corneal junction as has been maintained by Kruse. Scleral digitations are outlined by capillary loops. There is never any increase in the number of vessels composing the limbic plexus. Vessels certainly do cross the arcus senilis.

Few authors appear to have recognized that in the limbic plexus they are dealing with a physiologically very active capillary bed, as was so ably demonstrated ten years ago by Basil Graves in his studies on the vascularization of the cornea, observations which seem to have escaped the notice of practically everyone who has written on riboflavin deficiency. It is really only a matter of the extent to which vessels are open or closed. I have found that in order to study this problem with advantage it is essential to determine the reactivity of the vessels in this area by the use of capillario-dilator and -constrictor drugs.

By such means—and, so far as I know, no one else interested in riboflavin deficiency has used the method—it is possible to demonstrate a complete limbic plexus, for example, in an old man whose limbus on first examination appears to be avascular. On the other hand, one can satisfy oneself that a completely constricted and empty capillary becomes invisible. The method also serves to demonstrate telangiectasis formation, etc.

In regard to the actual vascularization of the cornea, my own experience confirms that of some others who have carried out surveys (Youmans *et al.*, 1942; Wilson, 1941; Kodicek and Yudkin, 1942; Pett, 1943; Tisdall *et al.* 1943; Wiehl and Kruse, 1941; Scarborough, 1942; Gregory, 1943; Sandstead, 1942; Machella and McDonald, 1943; Goldsmith, 1943; Keith Lyle

et al., 1944; Ferguson, 1944; Scott, 1944) in showing that it is not an unequivocal sign of ariboflavinosis; but that such a condition does not occur, as suggested by Vail and Ascher (1942), is incorrect. When these authors assert that—to use their own words—"Sydenstricker's pictures as well as his descriptions suggest that what he termed corneal vascularization is nothing more than extensive engorgement of the pre-existing limbal meshwork except in cases of obvious corneal disease," they display, I think, merely a lack of appreciation of the facts.

Another point I should like to make: my experience goes to show that it is quite impossible, owing to the great variability, to formulate any satisfactory method of notation for describing the degree of vascularity of the limbus and cornea, as has been attempted by some observers, including Keith Lyle and his colleagues (1944). No one seems to have commented on the individuality of the pattern of the intracorneal vascular network, which differs from that in vascularization associated with keratitis of inflammatory origin, etc. It is a point which may prove to have some significance.

Sydenstricker has drawn attention to the appearance of signs of ariboflavinosis in diabetes. The condition called rubeosis iridis—the subject of a communication by myself in 1942—its relationship to glaucoma, and the relationship of both to diabetes may here be mentioned.

As to the cause of the corneal vascularization, Bessey and Wolbach suggested it was of the nature of a compensatory process to bring blood into closer contact with the deeper layers of the corneal epithelium when the cornea is suffering from riboflavin deprivation. This explanation has been generally accepted in America and has found an adherent in Kruse, but to my mind it is no explanation at all and hardly consonant with Nature's methods. It has been assumed that riboflavin exists as such in the cornea and that it reaches the cornea by diffusing from the limbic plexus. Again, it has been held that riboflavin in the cornea is destroyed by light, as it is known that the pure substance is converted into an inactive form by ultra-violet irradiation. I must confess it appears to me strange that Nature should provide the cornea with riboflavin, there to be destroyed by light.

The work of Philpot and Pirie (1943) on the eye of the ox probably points much nearer to the truth of what obtains in man. They found that all the riboflavin present in the ocular tissues is in its coenzymatic form, which is unchanged by sunlight. They give the following values in microgrammes per gramme weight: for the substantia propria of the cornea, 0.2; ocular conjunctiva and corneal epithelium, 2.0; retina, 4.0; and, be it noted, lacrimal gland, 6.5; Meibomian gland, expressed secretion, 4.0. These authors pose the very pertinent question, Is it "possible that the riboflavin of the corneal epithelium is obtained from the lacrimal secretions rather than from the blood of the limbal loops"? Personally I feel that such a possibility would best fit the facts.

It appears to have been generally assumed that vascularization of the cornea is part and parcel of the same process as limbic injection. For my part I believe that with the earliest trophic changes in the cornea, due to riboflavin deprivation owing to a deficiency in the lacrimal secretions, proliferation-promoting intercellular hormones—to use the term adopted by Loofbourow (1942) for substances released by intact cells, when submitted to damage, into the surrounding intercellular fluid—are formed which stimulate the loops of the limbic plexus to throw out new vessels.

It would also offer an explanation of the occurrence of corneal vascularization in the vitamin-A-deficient rat which appears to be similar to, if not identical with, that associated with riboflavin deficiency. While Bessey and Wolbach referred to a change in permeability of the corneal epithelium as possibly being an important factor in causing vascularization in vitamin A deficiency, they looked to an interference with corneal respiration, and did not visualize, apparently, the possibility of that same change in permeability denying to the cornea its supply of riboflavin. The supply itself might be deficient, too, as the result of changes in the lacrimal and other glands. It is suggested, therefore, that the corneal vascularization in vitamin A deficiency may be a manifestation of a local riboflavin deprivation.

Corneal vascularization has also been described as associated with the following conditions, but further study will be necessary before their respective relationships are settled: (*a*) "Some unidentified component of vitamin B complex" (György, Echardt, and Johnson, *The Biological Action of Vitamins*). (*b*) Lysin and trytophan deficiency (Trotter and Day, 1942; Albanese, Randall and Holt, 1942, 1943). (*c*) Zinc deficiency (Follis *et al.*, 1942). (*d*) Sodium deficiency (Follis *et al.*, 1942). (*e*) Thallium deficiency (Buschke, 1913).

Neurological Symptoms

To the syndrome under discussion I admitted a group of neurological symptoms, all on the sensory side, chief among which were a sense of muscular weakness, incoordination, ataxia, and paraesthesia—unassociated with any real loss of power, any loss of sensation, or any muscular wasting—together with loss of visual and auditory acuity.

Though in the past these symptoms have been referred to by some authors as due to a peripheral neuritis, possibly influenced by ideas associated with beriberi, they have in fact no relation to those of that disease; nor have they anything in common with the peripheral neuritis described among alcoholic pellagrins in the United States, where the latter condition seems to have completely overshadowed all other neurological manifestations.

While I have suggested that these neurological symptoms, as in the case of other parts of the syndrome, are due to a hyporiboflavinosis, there is the possibility that some other factor may also be involved.

These neurological symptoms are of the same order whether associated with pellagra or not. They may be recognized in the accounts given by Scott and by Strachan of the neuritis in Jamaica, by Fitzgerald Moore in West Africa, by Landor and Pallister in Malaya, etc.; but for a fuller description I am going to the case of an English child, whom I had under my care some ten years ago, suffering from an acute recurrence of pellagra, exhibiting symptoms which we should now refer severally to nicotinic acid and riboflavin deficiencies. This child—incidentally, the first case of pellagra in this country ever known to have recovered—I was able to watch constantly day by day during a period of observation and during treatment, which was with yeast and liver extract. Nicotinic acid and riboflavin were then unknown.

The symptoms were entirely on the sensory side; together they formed a complete picture of the "cerebellar syndrome"—as that term has been used in its wider sense to include muscular weakness ("cerebellar asthenia"), muscular hypotonia, ataxia, asynergia, astasia, dysmetria, dysdiadokokinesis—together with nystagmus, vertigo, tremor, typical gait, and characteristic "pendulum" knee-jerk. At the same time there was some apparent mental apathy, hyper-emotionalism, as shown by the ease with which the child was moved to laughter or tears; and a facies somewhat reminiscent of Parkinsonism. Visual and auditory acuity were not tested. The most striking point about these symptoms was their changing measure, with rapid oscillations towards improvement or the reverse, suggesting fluctuations in the exciting cause, a reversible disorder of function. I am going to suggest that the disorder is located in the capillary bed serving certain areas in the central nervous system and that the symptoms are the expression of a metabolic disturbance in nervous tissue produced by the capillary dysergia resulting from hyporiboflavinosis. All those impulses serving the subconscious synergic regulation of voluntary co-ordinated movements, after relay, reach the cerebellar cortex. To them should be added those coming from Deiters's nucleus, the relay station of the vestibular nerve.

The cerebellar cortex is the receptive centre; co-ordination presumably takes place in the very rich synapse plexus of the molecular and granular layers; while the cerebellifugal fibres arise not in the cortex but from the intracerebellar nuclei, the chief of which is the dentate nucleus. On the other hand, after relay in the posterior horn, the fibres subserving skin sensation, pain, and temperature pass to the ventro-lateral nucleus of the thalamus. The thalamic nuclei are the centres for the correlation of sensory somatic impulses of all kinds before transmission to the cortex, whose activities probably enter into consciousness.

The initial reversible functional disorder, which of course if allowed to persist long enough leads to an irreversible lesion, is situated, I believe, in the synaptic structures of the cerebellum and is caused by a disturbance of its capillary blood supply. For the necessary facts I must turn to observations made on animals.

The metabolic activity of brain tissue is high; in the dog Craigie puts the oxygen consumption at 10% of the total for the whole body, whereas the brain weighs only 2% of the weight of the animal.

Capillarity of Nervous Tissues

The "capillarity"—that is to say, the relative number of capillaries in nervous tissues—varies very considerably in different areas and in different nervous-tissue elements. It is greatest in those areas and in those elements in which functional activity is greatest and metabolism highest. Those elements which have the greatest functional activity are the first to suffer the effects of any interference with their normal metabolic needs. As stated by Gasser (1937), the grey matter and the white matter of the central nervous system are differentiated by their metabolic needs. Greater vascularity, greater oxygen consumption, and greater susceptibility are characteristic of the centres as opposed to the fibres. This is not due, however, to the cellular content of the centres. The evidence goes to show that it is bound up with the mass of fine protoplasmic strands contributed by dendrites, terminal axon arborizations, and other synaptic structures, which together go to make up what Gasser refers to as the "neurophil"—a word which I think we would spell with a "y" instead of an "i," as it is presumably derived from the Greek φυλή=a clan.

For its proper functioning this system of fine strands of protoplasm demands a continuous expenditure of energy: lack of oxygen, etc., must soon bring about loss of function. In view of the fact that the surface area of a given volume of cytoplasm increases inversely as the diameter of the strands of which it is made up, it is obvious that the metabolism in the fine strands of neurophyl must be much higher than that in the cell bodies and axons.

Dunning and Wolff (1937) estimated (in the cat) the relative surface of the nerve-cell bodies, excluding the neurophyl, in relation to the length of capillaries per cubic millimetre, in the parietal cortex, the trigeminal ganglion, and the superior cervical ganglion, and showed that there was no correlation between the degree of vascularity and the cellular content of the grey matter. He inferred, therefore, that the correlation lay between the degree of vascularity and the neurophyl.

The actual degree of capillarity in various parts of the nervous system has also been determined by Craigie (1919, 1938). The *relative* average vascularity of different regions in the central nervous system of the rat is given below in units, adapted from Craigie's figures. He showed that the number of capillaries in the grey matter is greater than in the white matter. The least vascular grey matter is still one and a half times richer than the white matter. The vascularity of sensory nuclei is greater than that of the motor nuclei.

(*a*) Postero-lateral column (Burdach)	10
(*b*) Antero-lateral descending tract	12
(*c*) Antero-lateral ascending tract	14
(*d*) Pyramidal tract	20
(*e*) Postero-mesial column (Goll)	24
(*f*) Substantia gelatinosa Rolandi	34
(*g*) VIIth C.N. motor nucleus	42
(*h*) XIIth C.N. nucleus	46
(*i*) Vth C.N. motor nucleus	46
(*j*) Ventral horn	52
(*k*) Vth spinal nerve nucleus	53
(*l*) Deiters's nucleus	54
(*m*) Cerebellar cortex, molecular layer	56
(*n*) Dorsal horn	58
(*o*) Inferior olive	59
(*p*) Superior olive	64
(*q*) Vth C.N. sensory nucleus	64
(*r*) Cerebellar cortex, granular layer	68
(*s*) Nucleus dentatus	72
(*t*) VIIIth C.N. vestibular nucleus	78
(*u*) VIIIth C.N. cochlear nucleus	84

The most highly vascularized area in this set of observations was the dorsal cochlear nucleus (84), which is twice that of the facial motor nucleus (42) and eight times that of the fasciculus cuneatus (10). Nearly equal in vascularity to the cochlear nucleus comes the vestibular nucleus (78), followed by the dentate nucleus (72) and the granular layer of the cerebellar cortex (68). The relative position of the dorsal horn of the cord (58)—which includes Clarke's column—the molecular layer of the cerebellar cortex (56), and Deiters's nucleus (54) should also be remarked.

These figures for capillary vascularity appear to me most significant. They offer, I believe, the explanation of the localization of the group of the neurological symptoms that I have described. The cerebellar syndrome is due to a disorder of function of the cerebellar "neurophyl" as a result of being a tissue with high capillarity. I have not been able to find figures for the thalamus, but a similar disturbance in the thalamus might account for the sensations of "burning pain" without causing loss of skin sensibility. The emotionalism might perhaps similarly be explained; while a widespread but early functional upset in the cortex might produce the Parkinson-like condition and mental dullness. The figure for layer II of the occipital cortex probably approximates to that of the cochlear nucleus, followed in descending proportion by the parietal, temporal, precentral, and insular cortex.

This distribution of functional disorders I shall again refer to when I come to consider such pathological evidence as there is, but here two points may be added.

Oxygen consumption of cats' brain, estimated by the tissue-slice method, was found to be about the same in the parietal cortex as in the cervical sympathetic ganglion but seven times that of the trigeminal ganglion—confirming the view that metabolic activity lies in the neurophyl rather than in the cells. This correlation is also borne out by the work of E. Gordon Holmes (1930), who showed that the distribution of cytochrome and that of cytochrome oxidase run parallel to each other and to the rate of oxygen consumption.

The sites of the primarily functional lesion causing the loss of visual acuity and that causing loss of auditory acuity are uncertain, but I believe the lesions in the two conditions are of the same nature as elsewhere in the nervous system—i.e., a capillary dysergia. The high figure given for the capillarity of the cochlear nucleus is worthy of note, but there is none for the lateral geniculate body. Sydenstricker, who found the fundi normal, seems to suggest that the dimness of vision in his cases was due to the changes in the cornea; loss of auditory acuity he does not mention. It will be remembered, however, that Fitzgerald Moore also found a normal fundus in his early cases, but afterwards he ascribed the amblyopia to a retrobulbar neuritis as I had done, and of course later changes in the disk become obvious.

In the Spanish Civil War Grande Covian and Jiminez Garcia (1940) noted, among the syndromes they described, one which consisted of loss of visual and auditory acuity, commonly associated with the affection of the tongue and lips and neurological symptoms, including paraesthesiae and ataxia.

Impaired hearing was of course recorded by the older pellagrologists, as also were the ophthalmological conditions (Quaglino, Ferradas, Guaita, Denti, Rampoldi, Ottolenghi). During the present century really very few observations have been published (Whalley, 1909; Tucker, 1911; Ridlon, 1916; Calhoun, 1918; Jaensch, 1930; Krylov, 1932; Cronin, 1933; Levine, 1934; Carrol, 1936; Fine and Lachman, 1937; Laval, 1939; Mendoza Gonzáles, 1941; Alessandri *et al.*, 1942).

I think the evidence, so far as it goes, supports the idea that the condition in pellagra is essentially the same as that met with apart from pellagra and included in the hypo-riboflavinosis syndrome. I should prefer to call it a (retrobulbar) optic neuropathy.

The lack of systematization of the lesions in the central nervous system has generally been remarked (Kinnier Wilson, 1914; Greenfield and Holmes, 1939; Pauly and Deprecq, 1939), and remained unexplained up to date. There is a systematization, but it is one based on the neurophyl.

Pathology

The post-mortem findings in riboflavin deficiency in animals are not very conclusive in regard to the nervous system, but

on the whole would seem to lend support to the views expressed above (Street and Cowgill, 1939; Zimmerman, Cowgill, and Fox, 1937; Page, 1937; Wintrobe, Mitchell, and Kolb, 1938; Phillips and Engel, 1939).

There is no certainty concerning the pathological changes in the viscera in man in nicotinic acid and riboflavin deficiencies. It is just worth remembering, however, that degenerative changes occur in the heart muscle in pellagra, that the liver always shows fatty changes, most marked perhaps in infantile pellagra, while in the dog "yellow liver" is characteristic of ariboflavinosis. Scott mentions myocardial changes "with dilatation and congestion of the cardiac capillaries," and an angioma-like condition in the heart.

There is, so far as I know, no account of the post-mortem findings in human riboflavin deficiency. No case of "ariboflavinosis" has come to necropsy in America. None of the cases described by Fitzgerald Moore and by Landon and Pallister found its way to the pathologist. I have therefore turned to the detailed pathological records by Scott upon two cases of the Jamaican disease which he called "central neuritis" but which I have ventured to suggest, in the light of present-day knowledge, may have been acute hyporiboflavinosis. I wondered whether one would find a pathological picture in the central nervous system with irreversible tissue changes, such as might be expected to follow the early reversible functional disorder that I have supposed to occur. It is not possible to deal with these here in detail, but I believe Scott's findings support my thesis.

Scott noted the many points of resemblance between his own findings and those of Singer and Pollock (1913) in pellagra, who in turn had stated that the histopathological picture in pellagra was identical with that described by Adolf Meyer (1901) as "central neuritis." This condition, later referred to by Pearson (1920), I have little hesitation in suggesting is the later stage of the functional disorder I have described. It starts, I believe, in the neurophyl as the result of the capillary dysergia, and spreads to the cell, to be followed by changes in the axone.

The changes found in the pseudo-encephalitis of Wernicke associated with vitamin B_1 deficiency, consisting of capillary dilatation in certain areas of the nervous system—viz., the paraventricular grey matter of the third ventricle, the mammillary bodies, the peri-aqueductal region, the corpora quadrigemina, and the floor of the fourth ventricle—make an interesting comparison.

If, as I have suggested, riboflavin deficiency interferes with the normal functioning of the capillary, with the production of a tissue anoxia, it would seem of interest to compare the nervous symptoms so produced with those referable to the nervous system in chronic carbon monoxide poisoning.

C. K. Drinker in his monograph upon the subject states that headache occurs in 74%, dizziness in 50%, burning of the eyes in 32%, gastro-intestinal disturbances in 24%, drowsiness in 28%, cardiac disturbances in 18%, insomnia in 20%, faintness in 12%, neurological and mental symptoms in 17%. Among the last-named are giddiness, nervousness, emotionalism, tremors, muscular weakness, increased reflexes, neuromuscular pain, sensations of numbness and chilliness, paraesthesiae, ataxia, muscular incoordination, disorders of vision and hearing, neurotic and psychotic manifestations.

The general resemblance of these symptoms to those I have described in the syndrome here dealt with is, I think, worthy of note, especially when it is remembered that carbon monoxide and cyanide are believed to operate by inactivating cytochrome oxidase and thereby inhibiting oxygen consumption by preventing electron transfer—cyanide by uniting with the iron when in the "ferric" form, carbon monoxide when in the ferrous form. Of cyanide I shall not speak, but I might mention here that Quastel (1943) has recently analysed the evidence concerning the inhibition of oxygen uptake by narcotics, and concludes that flavoprotein is the sensitive factor.

Summary

An attempt has been made to disengage from the clinical affection we call pellagra a group of symptoms which may also be met with as a more or less complete syndrome apart from those symptoms generally recognized as due to a nicotinic acid deficiency.

This syndrome includes a good deal more than that to which American authors have given the name "ariboflavinosis." Any differences are, however, I think, a question of the comparative intensity and duration of the underlying pathogenic process.

I have considered the whole syndrome, for the sake of convenience, as due to riboflavin deficiency—a hypo-riboflavinosis I should prefer to call it rather than an a-riboflavinosis—but some uncertainty must still exist as to whether any other factor in the B, complex plays a part.

All the symptoms of the syndrome respond to treatment with yeast. It is suggested, in the absence of any knowledge on the subject, that we are warranted in making the assumption that the cells forming the capillary endothelium require all those elements needed by other cells in the body for their normal metabolism—sugar, oxygen, etc., including riboflavin.

It is further suggested that the first tissue to suffer the effects of riboflavin deficiency is the endothelium of the capillary system; that the initial result is a reversible functional disturbance of the capillaries—a "capillary dysergia"—which at the present time cannot be more accurately defined, but which is manifested by dilatation of these vessels and impaired flow; that in turn there is an interference with the normal cellular metabolism and in consequence a derangement of tissue function by disturbance in the *milieu intérieur*; and that the derangement of tissue function is relatively greatest in those tissues which possess the highest degree of capillarity and whose metabolism is greatest.

So long as the intensity of the functional disorder be not too great and so long as it do not persist too much, complete recovery may take place, otherwise irreversible processes occur which lead to pathological changes.

The hypothesis put forward would appear to offer an explanation of the incidence of symptoms and distribution of signs in riboflavin deficiency.

The interference with tissue-cell metabolism is probably of the nature of an anoxia in its wide sense.

It is suggested that disorders of capillary function—"dysergia"—quite apart from affections of the rest of the vascular system, may play a much more important part than has commonly been supposed in the pathogenesis of many conditions, including pellagra due to nicotinic acid deficiency.

At the risk of appearing mono-ideistic, I cannot help feeling there is perhaps a wide field awaiting investigation along such lines—a field embracing such widely separated conditions as fibrositic rheumatism, some psychoses, and neuropathies.

POST-OPERATIONAL STRAIN IN THE NAVY

BY

DESMOND CURRAN, M.B., F.R.C.P., D.P.M.

Temp. Surg. Capt., R.N.V.R.; Consultant in Psychological Medicine to the Royal Navy; Psychiatrist to St. George's Hospital, London

AND

GERALD GARMANY, M.B., D.P.M.

Temp. Surg. Lieut., R.N.V.R.; Neuropsychiatric Specialist

The concept of an operational strain syndrome is open to criticism on a variety of grounds. There is, for example, little evidence that psychiatric breakdowns associated with operational stress differ symptomatically from those that are not. Moreover, not all reactions supposedly associated with operational stress are due to it, and in wartime there is a very real danger of over-emphasizing the dramatic, such as exposure to enemy action, when less dramatic events, such as the regimentation and frustration of Service life, separation from home, and domestic difficulties, may really possess more significance. Again, the conception of an operational strain syndrome runs the grave risk of creating an honourable as opposed to a dishonourable or at least less honourable category.

There is, however, yet another objection, to which curiously little attention seems to have been paid—namely, that the concept is descriptively misleading in that most men break down after, rather than during, a period of stress; and it is with the phenomenon of post-operational strain that this paper is mainly concerned.

Discrepancy in Sickness Rate Afloat and Ashore

There are a number of observations which are not agreeable to the view that psychiatric casualties attributable to operational stress occur during operational stress. Thus, starting with the psychiatric problems as seen afloat, naval medical officers serving in ships have not hesitated to point out to naval psychiatrists serving ashore that, although at sea evidence of fear may exist in plenty, psychiatric cases do not. On the other hand, naval medical officers serving in naval establishments ashore, such as drafting depots, are keenly aware of the size and importance of the psychiatric problem in naval medicine, and this opinion is fortified by service in a naval hospital. That there is in fact a striking difference in the incidence of psychiatric disorders ashore and afloat is amply confirmed by calculations kindly made by Surg. Cmdr. J. A. Fraser Roberts, R.N.V.R., Consultant in Medical Statistics to the Navy, although for Service reasons it is not possible to publish the relevant figures at the present time. They can be up to ten times as numerous.

This discrepancy cannot be accounted for by the accumulation and concentration of the neurotic in naval depots ashore (e.g., in one naval depot 90% of psychiatric out-patients are passed fit for service at sea). Nor is the answer to be found in the ingenuously cynical view that men reporting sick ashore with psychiatric symptoms necessarily do so with any conscious or unconscious intention to evade their draft or their duty. Again, to see many of these men should convince anybody that they are not faking their symptoms.

Development of Post-operational Strain

It is submitted that *reduction* of "tempo" on coming ashore from sea is a potent factor not sufficiently recognized as a cause of symptom development. Men seem to develop a need for the stimulation to which they are accustomed, even if it is unpleasant. Alteration or reduction in the accustomed stimulation is a frequent cause of men reporting sick. It seems reasonable to regard the process as analogous, in slow motion, to the normal experience of feeling worse after danger than during it: the reaction after any excitement is often more disturbing than the excitement itself, especially if opportunities for a satisfactory outlet or distraction are lacking.

The state of physiological anxiety or tension is a condition of heightened responsiveness and preparedness, associated with well-known somatic accompaniments. At sea this increased responsiveness and readiness is not merely acquired passively by "conditioning," but is actively taught and fostered, both individually and collectively; for the safety of the group may depend on the degree of its development. Far from being pathological, it acquires the quality of a virtue which all should share, and is both necessary and completely appropriate. Some permanent degree of tension is thus established and maintained at sea as a biological necessity in order to cope with the emergencies that may at any moment arise; but after an initial relatively short-lived period of adaptation it obtrudes into consciousness only as the result of a special increase or decrement. A partial analogy may be drawn to the initial awareness of the noise of the dynamos on going on board, which then fail to be noticed until their cessation may once more call attention to their previous presence.

When a man goes ashore the increased tension that he has acquired at sea does not disappear immediately; the time required for it to do so varies considerably and is seldom less than a few weeks, although in some it may continue for months and even occasionally for years. During this individually variable period the increase of tension, which at sea was appropriate and necessary, is now incongruous. If a man starts violently at a sudden or loud noise he becomes aware of this incongruity, for sudden noises possess a much less ominous significance than they did. Further, he is aware of the discrepancy between his behaviour and that of his messmates, and he may become the object of their gibes. From the subjective standpoint, the increased tension that he has acquired is unpleasing and uncomfortable because it is inappropriate to the reduced tempo of his life; and objectively he shows the clinical signs of an anxiety state. The extent to which he will interpret his condition as evidence of an illness will depend upon his intelligence and personality and many other factors, and strong reinforcements may exist or develop to regard it as an illness, owing to unrecognized motives of an unworthy kind.

To recapitulate: Men at sea develop a condition of tension, resulting in increased responsiveness and readiness that are physiological and appropriate; but on coming ashore this tension state may become pathological, because it is inappropriate or unnecessarily intense having regard to the altered and reduced tempo of the life. The greater the lag that is shown in adjusting to the tempo that is suitable to the new environment, the more does the state that was previously within normal limits and physiological stand out as being abnormal and pathological. The basic reaction should be regarded as a psycho-biological occurrence, with a good deal of emphasis on the biological component, although more purely psychological determinants (of an impure or even unworthy kind, perhaps) may, and often do, come in secondarily.

Confirmation is readily forthcoming that the sequence of events described above is the correct one for many cases. Thus, when seeking for evidence of an objective kind about a patient's previous condition, it is not uncommon to hear surprise expressed by a shipmate that he should have fallen sick. There may be excellent testimony that the man was apparently normal and aroused no suspicion of disability on board, and equally good testimony of inefficiency and altered behaviour ashore. Again, men with first-class personalities not infrequently develop the symptoms of a severe anxiety state after admission to hospital for treatment for some incidental physical ailment. A careful history will also often disclose that the onset of anxiety symptoms coincided with in-from-sea leave. In this connexion it is noteworthy that the story of onset dating from a previous experience of enemy action is frequently quite inconsistent with the facts and is a retrospective falsification.

It may also be observed that certain men of a good type with persistent anxiety symptoms will ask to return to sea despite clinical signs that might appear prohibitive, and this endeavour to make the environment fit the state and to banish the sense of incongruity is often successful. The uneasiness of some combatant officers away from the front line and the desire of certain fighter pilots to spend their leave either spotting in the Channel or at sea in a destroyer appear to be of somewhat similar origin, and it detracts nothing from the courage of these gallant men to interpret their behaviour as being the expression of a biological need to sustain tension. The difficulties and breakdowns that occur in civilian life on retirement are perhaps examples of the same principle. Finally, the modicum of success which followed "conditioning" by providing psychiatric patients with some of the experiences of war (e.g., artificially produced sounds of dive bombers) may have been due to the manufacture of an artificial environment that was appropriate to the tension which these men experienced. It was soon recognized, however, that a return to duty was equally effective and more economical of manpower.

It seems plausible to postulate that reactive depression is often reactive to reactive anxiety. Reactive depressions preeminently occur in men of good personality after long periods of stress, and the affect is appropriate to a sense of having failed to run the course and of having succumbed to an illness bearing a stigma of shame. A belief of having been found wanting in "will power," a sense of impaired efficiency, lack of confidence, and a fear not so much of further enemy action as of experiencing again the tension that it produced, all contribute to the production of these states. The implicit teaching that underlies morale is such that few men fail to experience some sense of insult and guilt when pathological anxiety is experienced, and this may be why a measure of depression is so often mingled with anxiety states. This sense of guilt is less common in civilian life, and depressive accompaniments of anxiety states are also less frequent.

Prediction and Selection

The fact that those who finally succumb to stress form only a small minority would seem to constitute a weighty argu-

ment in favour of the rejection or early elimination of the vulnerable. There is no doubt whatever that those who do succumb show a high concentration of certain ominous points, such as a poor work record, compared with those who do not. According to certain investigations that have been made in the Navy, the proportion showing these ominous points amounted (with the technique used) to roughly 60% of psychiatric in-patients and 50% of psychiatric out-patients, as compared with only 15% among "normal" sailor controls. Yet naval psychiatrists keep 9 out of 10 of the men referred to them as out-patients at duty and send back to duty 2 out of 3 of their in-patients. Moreover, the relapse rate as judged by readmission to hospital does not seem unduly high. Thus, according to an investigation made last year, psychiatric cases were being returned to duty from hospital at about 5 times the rate that relapsed cases were being readmitted, and about half of these relapsed cases had remained outside hospital for more than 6 months. Finally, of those that are invalided for psychiatric disorders, two-thirds have survived in the Navy for over a year.

The significant thing about these findings seems to be that although a high proportion of those who break down not only give a past history which suggests their predictability as bad psychiatric bets, but also show that they were bad bets by having broken down, yet it is still possible to send the majority of them back to duty and to retain a relapse rate that seems reasonably small. This is, of course, not to doubt the desirability of selection procedures. The standard adopted for rejection or early elimination must naturally depend upon the available man-power; but a number do remain who should be rejected, no matter what the man-power position may be. It seems doubtful, however, whether in the Navy the proportion that should certainly have been rejected or eliminated amounted at most to more than 1 to 2%. This may seem a small number, yet the trouble these men cause is out of all proportion to their actual numbers, when considered in terms of hospital treatment, inefficiency, letting down others, and impairing morale. A great stand-by in the Navy has doubtless been the self-selection consequent upon men expressing a naval preference at the time of their call-up, for reluctant dragons do not make the best fighting material.

It seems reasonable to conclude that many "bad psychiatric bets" give very fair service before they break down and are also capable of giving further fair service after they have done so. These facts should counteract any undue pessimism that may exist as to the capacity of the neurotic to stand up to the stress of war. There can be little doubt that an additional factor inducing unwarranted pessimism is the abnormal or at least different situation that occurs in shore establishments, and particularly in hospital, compared with life afloat. The picture seen in hospital is especially misleading, and makes a correct assessment extremely difficult.

Treatment

Apart from the real, though limited, place that exists for pre-selection, probably the most that would be desirable prophylactically would be a warning to survivors and others going on leave from sea that anxiety would be experienced temporarily and should not be misinterpreted as illness. The vast majority of men, however, deal with their experiences quite adequately without outside help, and certainly any prophylactic "pep talks" of the above kind should be given by executive officers and not by medical men.

Once a pathological tension state has become established it must be dealt with either by reducing the tension or by raising the tempo. The latter can be accomplished by an early draft to sea before habituation, including doubts about future capacity, has occurred. Failure may result if the ship lies in harbour for too long a period.

Reduction of tension can be produced by sedation, and the barbiturates are eminently suitable for this purpose. Tremulous agitated patients should be put to bed, and 48 hours' sedation will make discharge to hospital unnecessary in the overwhelming majority. The average case seen as an out-patient in a naval depot shows a steady decrement of tension at a rate which is extraordinarily consistent. The administration of phenobarbitone in three doses of half a grain daily for one week, followed by the same dose twice daily for two weeks, usually brings about clinical recovery. With the other factors in treatment and management this paper is not primarily concerned. It may be worth noting that what medical men now effect with barbiturates humanity has accomplished for hundreds of years with the aid of alcohol. It is a sobering reflection that humanity was abreacting its pent-up emotions in parties with the aid of alcohol long before narco-analysis and group therapy were introduced for the same purpose.

"All theories are crutches, not sceptres or wizards' wands," and it is not suggested that the concept of post-operational strain that has been outlined is the whole story, but merely that it is perhaps a somewhat neglected part of it. Numerous other factors obviously help to make stress more tolerable and hence affect the incidence of neurosis, such as discipline, leave, mail, and the rapid solution of what are felt to be inequalities of treatment—to mention only a few. The Navy is, moreover, singularly fortunate in having a tangible object which is also a home around which group loyalty can centre—namely, the ship. An airman or member of the Tank Corps cannot feel about their contraptions in quite the same vividly personal way. Yet the life of a closed community, as in a ship, has its special dangers. Interesting browned-off (what the sailor call "chocker") paranoid reactions, rather similar to those seen among prisoners, may occur when the commission is too long or the ship is not a happy one. Dr. Johnson's dictum may be recalled: "No man will be a sailor who has contrivance enough to get himself into a jail; for being in a ship is being in a jail with the chance of being drowned." The rest of the quotation—"a man in jail has more room, better food and, commonly, better company"—is perhaps less acceptable.

There can also be little doubt that the group spirit and loyalty that exist on board ships are most potent factors in sustaining morale and that, conversely, the lack of this same group spirit and loyalty when a man leaves his ship and goes to a drafting depot is a most important factor in precipitating the breakdowns that occur there with so much greater frequency.

Summary

An endeavour has been made to show that psychiatric casualties attributable to operational stress occur most commonly after rather than during the stress.

Thus it has been pointed out that the psychiatric sickness rate is very much greater ashore than afloat.

The thesis is submitted that this is often due to a persistence of the physiological increase in tension developed at sea and rendered obtrusively uncomfortable by the suddenly reduced tempo on coming ashore; the reaction being analogous, in slow motion, to the normal experience of feeling worse after danger than during it.

While unworthy motives play an important part in perpetuation their role in producing the state can easily be over-estimated.

While pre-selection has a definite value, the practical importance of selection procedures is much modified by the fact that a substantial majority of psychiatric casualties can be returned to full duty.

Symptomatic relief often follows reduction of tension by sedation and increasing the "tempo" by early draft to sea before habituation has occurred.

SCHICK REACTIONS IN RECENTLY CONFINED WOMEN AND THEIR INFANTS

BY

G. PAYLING WRIGHT, D.M., F.R.C.P.

AND

W. M. CLARK

(From the Department of Pathology, Guy's Hospital Medical School)

It is now generally recognized that the resistance of newborn infants to diphtheria depends chiefly upon the passage of maternal antitoxin through the placenta during the latter part of gestation. So readily can this antibody enter the foetal circulation that at birth its concentrations in the serums of mother and infant are almost equal. From early childhood onwards any insufficiency in the concentration of circulating antitoxin to ensure immunity to clinical diphtheria may be readily demonstrated by a positive reaction in the Schick test

In very young infants, however, owing to certain physiological peculiarities in their skin, positive Schick reactions in susceptible individuals are much less readily evoked, and estimates of immunity obtained by the use of this test during the first few months of life are thus often fallacious. Consequently, the Schick reaction of the recently confined mother, which for practical purposes may be accepted as reflecting the antitoxin titres of both her own and her infant's blood, thus acquires a particular significance in the epidemiology of diphtheria in early infancy.

The following study was made during 1942 and 1943 in a maternity department of a hospital on the south-eastern outskirts of London. A large proportion of the 250 recently confined women examined came from districts in which housing conditions are good. Many of them, however, had spent the earlier part of their life as school-children in one of the Metropolitan boroughs of London, and, since it seemed possible that this might have had some effect upon their immunity as a group, the findings for these women have been recorded separately. The average age of the 250 women was 28.5 years. The Schick tests were carried out according to the usual technique 0 to 5 days after delivery. The sites of inoculation were examined daily for 7 days. The results of the tests are shown in Table I.

TABLE I.—*Schick Reactions (Totals and Percentages)*

Groups of Women Examined	+ve	ψ+ve	−ve	ψ−ve	Totals
London-bred ..	54 (45·7)	10 (8·5)	43 (36·4)	11 (9·4)	118
Others	75 (56·8)	5 (3·8)	45 (34·1)	7 (5·3)	132
Totals	129 (51·6)	15 (6·0)	88 (35·2)	18 (7·2)	250

Schick Reactions in Non-parturient Women

Since it seemed possible, from these rather high rates for positive reactions among recently confined women, that such persons might be more sensitive to diphtheria toxin in the puerperium than at other times, a further series of tests were made upon a group of 62 women who had been admitted to the same hospital for medical or surgical treatment. Their social surroundings were the same, and their average age—31.1 years—was nearly the same. Of this group 58% proved to be either positive or pseudo-positive reactors, so that there seems to be no reason to believe that the puerperal state gives rise to any heightened susceptibility to this toxin. The London-bred women in this group also showed a rather lower proportion of positive and pseudo-positive reactors (52.6%) than the remainder.

Positive Reactors in Recently Confined Women in Various Age Groups

In the group of 250 recently confined women there were statistically significant differences between the proportions of positive reactors in the following three subgroups: those 24 years and under; those between 25 and 34 years; and those 35 years and over. The numbers falling into these subgroups, the numbers showing positive or pseudo-positive reactions, and their respective percentages are set out in Table II.

TABLE II

Age Group	No. Tested	No. Positive	% Positive
24 years and under ..	88	60	68
25 to 34 years	116	67	58
35 years and over ..	46	17	37

Eighteen of these 250 recently confined women and 22 of the whole group of 312 stated that they had suffered from clinical diphtheria; 14 of them were among the 137 London-bred women—a proportion which might be expected from the ratio of notified cases to child population in the Metropolitan boroughs between 10 and 20 years ago (see Wright and Wright, 1942). Of these 22 women 12 reacted negatively to the Schick test, 7 positively, and 3 pseudo-positively. Only 5 of the 312 women had been immunized prophylactically against diphtheria; 4 of them had a negative and one had a pseudo-positive reaction.

Changes in Schick Reactions at Short Intervals after Injection of Toxoid

It appeared unlikely that so large a proportion of women living in a semi-urban environment could have escaped immunization through casual contact with diphtheria cases or carriers at some period of their lives. It seemed much more probable that most of them had undergone some degree of immunization in the past, but that the exposure was too slight or at too remote a date to maintain their circulating antitoxin titre at or above the critical level for the Schick test. To examine this possibility some of the positive reactors were injected intramuscularly with 1 c.cm. of T.A.F. on the third or fourth day after their Schick test. A second Schick test, in which toxin alone was used, was carried out four or six days after this immunization. The diameters of the hyperaemic areas present at the peak of the responses were measured, and in most instances the relative intensities of the first and second reactions were noted roughly for comparison. With the help of this combination—the comparatively large "recall dose" of toxoid and the two Schick tests—it was hoped to distinguish those women in the two subgroups: (*a*) "fully susceptible" and "sub-immune," from (*b*) "positive immune" and "potentially immune" (see Glenny, 1925).

Of the two subgroups of 20 women each whose tests were repeated 4 and 6 days after receiving the "recall dose" of toxoid, 10 and 9 respectively showed second reactions two-thirds or less the size of those originally found. In the 6-day group, possibly owing to the rather longer interval, the intensity of the hyperaemia in the second test was in almost all instances notably less than in the first. Such changes in the size and intensity of the reaction almost certainly indicate that the second injection of toxin had been more fully neutralized than the first, and consequently that an appreciable amount of antitoxin had been formed actively by such women—a response to this antigenic stimulus that would be unlikely to take place so promptly except in the tissues of an already partially immunized person.

From these observations it seems likely that at least half, and perhaps more, of the positive reactors among the recently confined women fell into the categories termed by Glenny the "positive immune" and the "potentially immune." At some time in the past they would probably have reacted negatively to this test, but, possibly owing to long residence in less densely populated districts, they have relapsed into the positive state.

Schick Reactions in the Infants

Schick tests were also carried out at some time during the first 5 days of life upon the infants of 145 of this group of women. The injections were made either in the scapular region or on the outer aspect of the forearm near the elbow. All the test sites were examined daily for 7 days, except in a few instances in which the mother or infant had left the hospital on the fifth or sixth day. In the infants no reaction was regarded as positive unless it was seen to be present on at least two such examinations. The findings in the present series, together with those of certain earlier workers, are shown in Table III.

TABLE III

	Infants All Under	Mother +ve Infant +ve	Mother −ve Infant −ve	Mother +ve Infant −ve	Mother −ve Infant +ve
Ruh and McClelland (1923)	7 days	20	75	4	1
Kuttner and Ratner (1923)	14 „	4	39	7	0
Cooke and Sharma (1932)	10 „	59	204	34	1
Greengard (1932)	14 „	22	45	33	0
		Agreement		Disagreement	
Totals .. .. Percentages ..		468 85·4		80 14·6	
Present series ..	5 days	66	56	21	2
Totals .. .. Percentages ..		122 84·1		23 15·9	

In 122 of the 145 mothers and infants tested their reactions agreed. In the remaining 23 there was disagreement—the mother being negative and the infant positive in 2 instances, and the reverse in 21 instances. The circumstances under which these disagreements occurred were as follows:

Mother Negative and Infant Positive (2 instances).—In the first of these the mother was pseudo-negative and the infant reacted only faintly on the second and third days. In the other the woman was definitely negative and the infant definitely positive. It may be observed that instances are recorded in which a relatively high antitoxin titre in the maternal blood is accompanied by a much lower, or even untitratably low, level in the cord blood. Von Groër and Kassowitz (1915) found three such instances in their series.

Mother Positive and Infant Negative (21 instances).—In 6 of these instances the mothers were definitely positive. Three of their infants showed a positive reaction on one day only, and were accordingly classified as negative; in one other instance the mother had had diphtheria 24 years before. In 5 other instances the mother was classified as a faint reactor, and in none did the infant show any sign of a positive reaction for even so short a period as 24 hours. In the remaining 10 instances the mothers, three of whom had had diphtheria in childhood, showed pseudo-positive reactions. One infant in this group showed a positive reaction on one day only.

The most common form of disagreement was thus a failure of the infant to react positively when the mother was positive, and especially when she was either a faint or a pseudo-positive reactor. This state of anergy was present in about one-quarter of the infants in whom a positive reaction might have been expected from the result of the test upon the mother. The proportion is rather smaller than that found in the earlier studies, and is probably due to the frequent examination of the test sites in the present series. Moreover, the lighting conditions for examining the present group of infants were unusually favourable.

Discussion

The belief is still widely held that the great majority of young infants possess a high degree of congenitally acquired passive immunity to diphtheria for some months after birth. Two circumstances in particular have contributed to this belief. First, many of the observations upon specific immunity in newly born infants were made in Vienna and other European cities, in whose populations diphtheria was particularly prevalent, and the results of these early surveys have been applied rather freely to other places and times without allowing adequately for local differences in the endemicity of the disease. Secondly, insufficient attention has been paid to the phenomenon of skin anergy which is shown by young infants to diphtheria toxin and other noxious agents capable of producing delayed erythematous reactions, so that, for this age group, many negative Schick reactions have been interpreted erroneously in consequence. Together, these misconceptions have led to an exaggerated estimate of the prevalence of congenital immunity during the first few months of life, and the reliance which has been placed upon this supposed insusceptibility is unfounded in a large proportion of instances.

The present observations indicate that considerably less than half of the infants born to women living in a typical outer London suburb have any adequate congenitally acquired immunity to diphtheria. So infrequently do the mothers come into contact with toxigenic *C. diphtheriae* that their circulating antitoxin concentrations fall to levels considerably below the critical titre for the Schick test. While a large proportion of the positive reactors among these recently confined women are themselves immune in consequence of the promptitude with which their antitoxin-forming tissues respond to the secondary stimulus of the specific toxin, their low antitoxin titre during pregnancy confers little or no immunity upon their infants.

At the present time the trend of mortality of infants under 1 year from diphtheria in England and Wales is a declining one, and so long as this fall continues no additional prophylactic precautions seem to be required. In the event of an epidemic rise, however, either in some particular locality or in the country as a whole, two possible measures might be instituted. First, infants might be immunized considerably younger than is now the custom: this procedure has the disadvantage that very young infants seem to respond less well to prophylactic immunization than older infants or young children. Secondly, as Dudley (1934) suggested, it might be desirable to immunize all Schick-positive pregnant women during the latter part of the gestation period. Such a procedure would at the same time immunize any woman who was susceptible and raise the maternal antitoxin titre to a high level about the time of the birth of the infant.

REFERENCES

Cooke, J. V., and Sharma, B. M. (1932). *Amer. J. Dis. Child.*, **44**, 40.
Dudley, S. F. (1934). Med. Res. Cncl. Sp. Rep. Ser. No. 195, London.
Glenny, A. T. (1925). *J. Hyg.*, Camb., **24**, 301.
Greengard, J. (1932). *Arch. Pediat.*, **49**, 526.
Kuttner, A., and Ratner, B. (1923). *Amer. J. Dis. Child.*, **25**, 420.
Ruh, H. O., and McClelland, J. E. (1923). Ibid., **25**, 59.
Von Groër, F., and Kassowitz, K. (1915). *Z. ImmunForsch.*, **23**, 108.
Wright, G. Payling, and Wright, H. P. (1942). *J. Hyg.*, Camb., **42**, 451.

RADIOTHERAPY OF ECTOPIC CALCIFICATION

BY

E. MILLINGTON, D.M.R.

Radiotherapist to the Royal West Sussex Hospital, Chichester

For some years radiotherapists have been treating with success deposits of calcium in the tissues, but unfortunately few outside the specialty seem to know of them. In two recent books on bone and joint conditions (McMurray, 1937; Watson-Jones, 1943) no mention is made of radiotherapy at all in this connexion, and few writers on general surgery have commented on this mode of approach, although in America the use of *x* rays in treatment is much more widely appreciated, and standard works, such as that by Comroe (1941), give full credit to radiotherapy. Ectopic calcium deposits may appear almost anywhere in the body, but of most interest here are those occurring in muscles, tendons, and bursae, as such deposits are in many cases painful and thus attract attention and demand treatment. The shoulder region is often affected, as in the calcified subacromial bursa or supraspinatous insertion, and the olecranon and Achilles bursae are also quite often involved in this process.

No convincing explanation of this deposition has as yet been given, and the formation of actual bone is even more obscure. Many theories have been put forward on the subject; these are summarized by McCurrich and Millington (1943). It is only with calcification, and not with true ossification, that we are now concerned, as *x*-ray treatment has no effect on formed bone, but only on calcium deposits. Calcium is frequently deposited in dead or devitalized tissue, possibly because of the change in *p*H in such regions, and the lesions with which we are dealing are mostly traumatic in origin, with pain dating from some injury or strain and becoming more persistent with time and the usual methods of treatment. *X*-ray examination eventually shows the presence of shadows due to the deposition of calcium of an amorphous and flocculent nature. It seems questionable whether it is the calcium that causes the pain, but in most cases if the calcium can be removed the pain also goes to a large extent, and perseverance with physiotherapy will then complete the cure.

Many surgeons recommend and practise surgical removal of the calcium. It was sometimes found at operation, however, that what appeared radiographically to be a large mass of chalk could not be distinguished from surrounding tissues when exposed, and at other times only a small amount of chalky fluid was seen; so there is no guarantee of cure from operation. *X*-ray treatment offers a method which is devoid of trauma and risk, and in any case does not preclude any more drastic methods later should it fail. The mode of action of the rays is not known, any more than it is with other applications; it may be due to alteration of blood supply, or quite possibly to some change in the *p*H of the irradiated part. The dosage is small and not harmful, there are no known ill effects and the patient is not laid up or incapacitated in any way; but it must be realized that the effects of *x* rays are not usually manifest for some time after the treatment has been given, and as a rule it is some four to six weeks before there is any change in the radiographic appearances even if the pain has been relieved earlier.

Technique

The technique of treatment is not standardized, and good results are obtained by widely varying methods, but one technique which has proved satisfactory in the hands of quite a number of therapists employs repeated small doses of highly filtered *x* rays—namely, 200 *r* at 200 kV with 0.5 mm. Cu and 1 mm. Al filter, given once a week for four doses. Re-examination of the part after four weeks will often show absorption of most of the calcium; if not, the course may be repeated. Only a small area is treated, and there should not be any skin reaction or adverse systemic effect.

Three typical cases are reported below—one occurred in the subacromial bursa, one in the tendo Achillis bursa, and one in the soft tissues of the hand. All were of long standing, and with treatment all cleared up completely, both radiologically and clinically, although the cause was quite different in each case.

Illustrative Cases

Case 1.—An area of calcification was found under the deltoid on *x*-ray examination of this patient, who had had pain and stiffness in the shoulder for five years after falling from a horse. The pain had become continuous and had been accentuated by a course of radiant heat administered by the patient to himself. A single course of treatment was given as above, with relief of pain two days after the first dose, and no return to date (nine months). Radiographs taken four weeks after treatment showed no definite opacity; only some scattered mottling was seen.

Case 2.—A bursitis of the tendo Achillis region followed injury to the heel 12 months ago in this patient, suspected of gonococcal infection. Radiographs showed a definite patch of calcification, and a small sinus formed which discharged a creamy fluid at intervals. After one course of treatment the pain was much easier; but the discharge continued, and radiographs at this stage revealed considerable diminution in size of the patch of calcium, but not complete regression. A second course was therefore given, after which the discharge stopped, pain vanished, and radiographic examination showed complete disappearance of the deposit.

Case 3.—Localized deposits of calcium were found in the hand of this patient, who is suffering from a very low-grade type of Hodgkin's disease. The deposits were palpable and very painful, with some oedema of the surrounding tissues. An attempt was made at surgical removal, but this was unsuccessful, as no discrete deposit was found on exposure of the region involved. After one course the pain had gone and radiographs showed almost complete disappearance of the calcium, so no more treatment was given. The patient is well to date, after one year, but has grown other deposits elsewhere which have not yet been treated.

Comment

In these three examples ossification had not occurred, and it must be emphasized that radiotherapy is efficacious only when unorganized calcium is present; but, nevertheless, in early ossifying lesions *x*-ray treatment will often prevent the formation of bone from the preliminary calcium deposits. In the elbow region a deposition of calcium occurs before ossification in the antecubital muscles, and in the early stages will disappear if treated with radiation; some American surgeons even recommend the prophylactic irradiation of elbow injuries to prevent subsequent myositis ossificans. Haematomata may be treated in the same way with small doses of *x* rays and ossification be prevented in most cases. Unfortunately, organized deposits of calcium in the form of biliary and urinary calculi show no response to doses of *x* rays.

References

Comroe, B. I. (1941). *Arthritis and Allied Conditions*, Philadelphia.
McCurrich, H. J., and Millington, E. (1943). *Brit. J. Surg.*, **31**, 86.
McMurray, T. P. (1937). *Practice of Orthopaedic Surgery*, London.
Watson-Jones, R. (1943). *Fractures and Other Bone and Joint Injuries*, Livingstone, Edinburgh.

A post-war programme of measures to reduce road accidents has been published by the Pedestrians' Association (180, Fleet Street, E.C.4). The proposals include better speed control, stricter enforcement of the law, a higher standard of efficiency among drivers, and a safety motor car. The Association takes the view that the 30 m.p.h. in built-up areas is too high a limit for safety and that the limit should be 25 m.p.h. with higher speed limits on main roads outside the built-up areas. Proposals for a safety car include placing the engine at the rear, giving the driver a better view of the road; greater manœuvrability; a shock-absorbent frame; and flexible upright fenders in front of the leading wheels with a net catcher between them.

Medical Memoranda

General Hypersensitivity to Sulphanilamide after Surface Application

Although the occasional development of general hypersensitivity to sulphathiazole after the application of that drug to skin lesions has been described (*J. Amer. med. Ass.*, 1943, **121**, 406), no mention is made in the Medical Research Council's War Memorandum No. 10 (The Medical Use of Sulphonamides) of a similar occurrence with sulphanilamide. The following case may therefore be of interest.

A telegraphist aged 21 complained on March 8, 1944, of a sore throat of 24 hours' duration. His temperature was 101.6°, pulse 100, and respirations 20. The tonsillar lymph glands were palpable and tender, both tonsils were slightly swollen, and the fauces and oropharynx were injected. He was given 2 g. of sulphanilamide at 8 p.m., to be followed by 1 g. 4-hourly. Next morning his temperature had fallen to 97.4° and his throat felt much better. At 8 p.m., however, 24 hours after sulphanilamide therapy had been started, his total intake being 8 g., the temperature was 103° and a generalized skin rash had developed. It was most marked on the dorsal surfaces of the hands, on the forearms, and on the feet and legs; and it was morbilliform in character, except on the hands, where there were some clear vesicles about the size of a pin-head. The throat was only a little sore and injected. No more sulphanilamide was given and the temperature fell abruptly. It was below normal on the 10th, but was raised, up to 102°, on the following 4 days. The rash faded slowly, and on March 13 it was rather more urticarial than morbilliform. Its distribution had also changed, for it was almost as intense on the front of the chest as on the hands and feet. Ten days later the only abnormality was slight scaling of the skin of the hands, forearms, ankles, and legs, which persisted for a further 3 weeks.

It was found from his previous records that sulphanilamide paste had been applied to an incised wound, about 2 in. long, on the front of the right leg, from Oct. 9 to 12, 1943—i.e., for four days—five months before this illness. The only other occasion on which he had received medical attention since he was a child was in 1940, when he had a cough, for which he was given a "black bottle." Thus there seems to be little doubt that the appearance of pyrexia and a rash after only 24 hours' treatment was due to sensitization by the surface application of the drug.

G. G. WALLIS, M.B., B.S.,
Surg. Lieut., R.N.

Weil's Disease without Jaundice

The name Weil's disease has been criticized on the ground that Weil was not the first person to describe the disease, but alternatives such as spirochaetosis icterohaemorrhagica and spirochaetal jaundice, which lay so much stress on the jaundice, probably result in many cases being misdiagnosed. We have recently had four consecutive cases in which there was no jaundice.

Four Recent Cases

Case 1.—A child aged 4 fell into a brook which was flooded with storm water. The accident occurred in the winter, and was followed immediately by a brief febrile illness which was diagnosed as pulmonary collapse and treated with sulphathiazole. Chest symptoms and signs soon disappeared, but the child remained unwell and refused to eat. On the eleventh day she vomited, and there was an abrupt rise in the temperature to 105°. On admission to hospital two days later her temperature was 103°, her conjunctivae were injected, and there was a trace of albumin in the urine. The serum agglutinated *Leptospira icterohaemorrhagiae*. The acute symptoms rapidly subsided; no other physical signs appeared, and the urine was normal after the first day. Nevertheless, it was some months before the child was really well again, and on several occasions there was a slight rise in the evening temperature.

Case 2.—This case was discovered accidentally after a short febrile illness originally diagnosed as influenza. The patient was a young doctor who worked in one of the Emergency Public Health Service laboratories. He described an abrupt onset of malaise with fever, pains in the limbs, headache, and sore eyes. After six days he had apparently recovered, but realized he still lacked energy. He therefore continued to take his temperature, and a fortnight after the onset of his illness he found it was 100° on two consecutive evenings. The head of his department, who knew there was no epidemic of influenza at the time, insisted on routine agglutination tests on his serum, and the diagnosis of Weil's disease was made in this way. The patient had bathed in the Cherwell four weeks before the illness began—probably too long an interval for infection to have occurred then—but his home was near the river, and rats had been seen there. At this stage there were no objective signs of ill-health, but he was still very easily tired, and it was nearly three months before he felt really fit.

Case 3.—This patient had also been bathing in the Cherwell. He was sent from a boys' harvest camp with a provisional diagnosis of poliomyelitis. Four days before admission he suddenly developed a severe headache and pains in the legs, and on admission his

temperature was 101°, his eyes were inflamed, and there was albuminuria; the calf and thigh muscles were extremely tender, and the deep reflexes were absent. Weil's disease was immediately suspected, and the blood urea was found to be 74 mg. per 100 ml. For four days he was very ill, but then rapidly became convalescent. The albuminuria disappeared and the blood urea fell to normal; but the sedimentation rate, which was 54 mm. per hour on admission, rose to 96 and remained high. For the next four weeks he felt perfectly well so long as he was kept in bed, but on the 40th day there was a transient recrudescence of fever accompanied by albuminuria. At no stage was he jaundiced, nor did the plasma bilirubin rise above 0.4 mg. per 100 ml.

Case 4.—The fourth patient was an airman, aged 22, who was scratched on the leg by a rat. Ten days later he suddenly felt feverish; he complained of headache and pains in the limbs and began to vomit. His temperature was 102°. The fever and symptoms persisted for seven days, when he was admitted to hospital. On admission his temperature was 101° and the conjunctivae were injected; there were no other physical signs and no albuminuria. Three days later he was afebrile and felt much better, but two weeks afterwards he suddenly developed mistiness of vision and was found to have punctate keratitis.

COMMENT

In each of these four cases the diagnosis was confirmed by agglutination of the *Leptospira icterohaemorrhagiae* by the serum in a titre of 1 in 1600 or higher. Since then we have had a fatal case, with intense jaundice and uraemia, in an Italian prisoner who was employed in draining fields. Hitherto the number of non-icteric cases has been estimated at approximately 50%, though Kramer (1934) of Rotterdam reported 73 cases, of which only 28 developed jaundice, and in cases in which meningitis develops jaundice is more commonly absent than present (Lescher, 1944). The low incidence of jaundice in our series suggests that in the past many non-icteric cases have been missed. We think this would be less likely to happen if the name spirochaetal jaundice was not so misleading and if it was not too exclusively regarded as an occupational disease. Weil's disease should be suspected whenever the sudden onset of fever is associated with headache, pains in the limbs, and conjunctivitis. Albuminuria, a rise in the blood urea, and a high sedimentation rate are also characteristic. Once the cause is suspected the diagnosis is easily confirmed by the specific agglutination reaction. Our cases support the saying, "No jaundice, no death," but emphasize the importance of keeping the patient under observation for several weeks and allowing a prolonged convalescence.

ALICE STEWART, D.M.
L. J. WITTS, D.M., F.R.C.P.
Radcliffe Infirmary, Oxford.

REFERENCES

Kramer, P. H. (1934). *Nederl. Tijdschr. Geneesk.*, **1**, 1280.
Lescher, F. G. (1944). *British Medical Journal*, **1**, 554.

A Case of Fat Embolism

We report a case of fat embolism presenting the unusual features of a delayed onset after operation without any of the premonitory chest signs.

CASE HISTORY

The patient was a prisoner of war, aged 33. In Aug., 1941, he sustained a tangential bomb wound of his right humerus, with a fracture at the junction of the middle and lower thirds. He was admitted to a military hospital on Oct. 5, 1943, with complete non-union of the fracture. There was a well-healed scar on the outer side of his arm, with generalized muscle-wasting, but no evidence of nerve injury.

A bone-graft operation was performed on Oct. 12. The bone ends were freshened and a 4-in. intramedullary bone peg from the left tibia was introduced. A complete shoulder spica was applied. At the close of the operation the patient was somewhat shocked, and one pint of fresh blood was transfused. The early post-operative progress was satisfactory. After 12 days he complained of bleeding from his gums and there was purplish bruising round his left knee and ankle. He was given vitamin C; the stitches were removed from his left leg, which was well healed, and he started to get up and sit in the sun.

Three days later (Oct. 27) he was noisy and irrational at night and incontinent of urine: temperature 98°; pulse 80; no cough, dyspnoea, or cyanosis. The stitches were removed from the right arm via a window in the plaster. The wound healed cleanly, though bruising round the left knee and ankle was more marked. Knee- and ankle-jerks were exaggerated; plantar response was doubtful. There was no evidence of any paralysis. The optic disks appeared normal. Intravenous glucose-saline given. Urine: albumin +; sugar + + + ; ketones + ; fat globules present. Blood sugar, 170 mg. per 100 c.cm.

Next day (Oct. 28) his general condition had deteriorated: temperature 97°; pulse 88. He was kept tranquil only with luminal and morphine. The plaster spica was removed from the back. Examination of the chest revealed moist crepitations at bases only; no sign of consolidation. On the 30th there was a slight improvement. He was quiet all day without a sedative. On Oct. 31 he was rational. Drip saline was discontinued after infusion of 36 pints in 3½ days. His condition then rapidly improved. A new plaster spica was applied on Nov. 2, and on Nov. 3 he started to get up.

Laboratory Investigations.—The blood and urine examinations are best demonstrated in a table, as follows:

Date	Blood Sugar (mg. per 100 c.cm.)	Urine					
		Albumin	Sugar	Ketones	Acetone	Fat	Casts, Granular and Hyaline
27/10/43	170	+	+ + + +	+	−	+ + +	−
28/10/43	160	+	+ + + +	+	−	Not tested	−
29/10/43	150	+	+	+	−	,,	−
31/10/43	140	+	Trace	−	−	,,	−
1/11/43	140	+	,,	−	−	,,	−
3/11/43	100	+	−	−	−	−	+ +
4/11/43	100	+	−	−	−	−	−
13/11/43		Trace	−	−	−	−	+
16/11/43		−	−	−	−	−	−

The tests employed were: (1) Benedict's test for sugar in the urine. (2) Crocelius-Seifert picric acid method for blood-sugar estimation. (3) Fat in the urine was demonstrated by staining with Sudan III. Globules of fat varied in size from a few to 30–40 microns in diameter. It was also demonstrated indirectly by the "sizzling test." In this test, adopted by Major J. V. Wilson, R.A.M.C., a loopful of fat-containing urine is burnt in a spirit flame and makes a distinct sizzling, which is very striking compared with that of normal urine. (4) No sputum was available for fat-globule staining.

COMMENT

The glycosuria and blood-sugar level cannot be entirely explained by the intravenous glucose-saline, because, while this was maintained approximately constant, the blood sugar and glycosuria progressively diminished. Also, the glucose was given at the rate of 0.12 g. per kg. of body weight per hour, whereas in the experimental animal it can be injected at a rate of 0.9 g. per kg. per hour before it appears in the urine. The glycosuria would therefore seem to be, at least in part, of renal origin.

Evidence of renal damage is afforded by the presence of albuminuria and casts, and may be assumed to be due to fat embolism of the kidneys. Glycosuria might thus have resulted from a selective renal damage comparable to that caused by phloridzin. If it occurs with any constancy in fat embolism it might be a point of diagnostic significance. It was observed in another case of fat embolism occurring in 2 N.Z.C.C.S. in the Western Desert in Nov., 1942, which at necropsy was proved to have extensive fat embolism of the kidneys and other organs. Albuminuria and ketonuria were present, but the clinical features and other circumstances of the case differed considerably from the one under review.

We are indebted to Lieut.-Col. A. Cruickshank, R.A.M.C., for permission to publish this case.

G. ST. J. HALLETT, F.R.C.S., Capt., R.A.M.C.
A. J. M. DRENNAN, M.B., Capt., R.A.M.C.

Torsion of the Gall-bladder

The rarity of the above condition, I hope, warrants its publication; it is the first such case that I have seen in over 30 years of practice.

Miss A., aged 67, in normal health, was reaching up to put a shilling in a slot meter on Feb. 4 when she was seized with acute abdominal pain under the right costal margin. She was not sick and thought it might be appendix trouble, so she starved herself to some extent as her own means of treatment. The pain persisted, and on Feb. 7 she sent for her doctor. At this time the patient had no rise of temperature or pulse rate, and no sickness, but her pain was severe, and a tender lump was palpable below the costal margin.

I was asked to see her on Feb. 8 and found the conditions as above. The patient was transferred to a nursing home on the 9th, and Dr. Shera, the pathologist, kindly did a white count for me, which showed 6,800 leucocytes only. On Feb. 10 I performed a laparotomy, making a Kocher's gall-bladder incision. The transverse colon was adherent to the liver edge; this separated readily with a gauze swab, and immediately the gall-bladder came into the wound with a long pedicle which untwisted easily. It had been lying in a bed formed by coagulated lymph and exudate. I removed the gall-bladder, ligatured the cystic duct with catgut, drained the cavity, and closed the abdomen. The gall-bladder was found to have two medium-sized stones in it, and was tense and full of almost pure blood. There was no infection.

The patient made an uninterrupted recovery, and went home on March 8.

I am greatly indebted to Dr. Downing for allowing me to publish this case, and to Dr. Shera for the blood count.

Eastbourne. E. WILSON HALL

Reviews

RECORD OF MEDICAL PROGRESS

The British Encyclopaedia of Medical Practice, including Medicine, Surgery, Obstetrics and Gynaecology, and Other Special Subjects. Medical Progress, 1943 and 1944. Under the general editorship of Sir H. Rolleston, Bt. (Price 37s. 6d. each volume, postage extra.) London: Butterworth and Co.

Having completed the onerous task of editing the *British Encyclopaedia of Medical Practice* Sir Humphry Rolleston continues to issue annual supplementary volumes under the title of *Medical Progress*, the fourth and fifth of which lie before us. The general plan is to divide each volume into three parts: the first composed of three critical general surveys with special articles on subjects of topical interest; the second on recent developments in drug therapy from the pen of Prof. W. J. Dilling; while the third, comprising about three-quarters of the whole, is devoted to abstracts alphabetically arranged. This method, though convenient for reference, inevitably leads to repetition. Thus, in the 1943 volume the Rh factor is dealt with in four articles, and as each writer explains the facts in a slightly different way the inexpert reader may be left rather confused. But of the general value and helpfulness of these volumes there can be no doubt. One is left with a lively sense of our indebtedness to the organic chemist in unravelling the complexity of the chemistry of the steroids and vitamins, and for the rapid multiplication of varieties of sulphonamides adapted to different diseases. Quite appropriately war medicine and surgery bulk large, to which Sir Arthur MacNalty contributes a valuable article on public health in the present war. It is perhaps invidious to try to pick out individual articles, but we were specially attracted by Dr. A. W. Spence on endocrinology and Dr. Oakley on diabetes. On thymectomy for myasthenia gravis a very cautious attitude is taken, but we were interested to read that W. G. Millar and T. F. Ross, while inquiring into "thymic death," established a significantly higher degree of aortic hypoplasia in cases of fatal accident than in the controls. Does this confirm the belief of insurance companies that some individuals are naturally "accident-prone"?

A VIEW OF RHEUMATIC FEVER AND CHRONIC ARTHRITIS

Pathology and Therapy of Rheumatic Fever. By Leopold Lichtwitz, M.D. Foreword by William J. Maloney, M.D., LL.D., F.R.S.Ed. Edited by Major William Chester, M.C. (Pp. 211; illustrated. $4.75.) New York: Grune and Stratton. 1944.

Prof. Lichtwitz, recognized as an authority on the pathology of function, completed this book only a few weeks before his death. His main thesis is that the mechanism of rheumatic fever is allergic sensitization of the mesenchymatous tissues, especially the vascular system in its smaller and smallest branches, and he has put forward a strong case supported by scientific and clinical evidence. He states dogmatically that rheumatism is caused by a sensitization to antigens, protein in nature, usually the product of micro-organisms which may be pathogenic or non-pathogenic, but may also arise in response to foreign proteins which are not of microbic origin. Although the book bears the title Rheumatic Fever, the author includes chronic arthritis, which in many forms he regards as of the same nature, but he draws a distinction between rheumatic and non-rheumatic chronic arthritis; the latter he is inclined to consider as a systemic disease with involvement of the central nervous system, especially the vegetative centres. His net is indeed cast so widely that many diseases not usually regarded as rheumatic are brought within it, such as dermatomyositis and lupus erythematosus.

The discussion of some of the chronic rheumatic diseases is of interest and often provocative, possibly because Lichtwitz's method of classification cuts directly across the more usually accepted systems. His description of the types and clinical features of spondylarthritis and also of skeletal lesions will provoke criticism, and other examples might be instanced. There seems at times to be a tendency to fit the symptoms to the theory, instead of the reverse. Dr. Maloney, who contributes an interesting foreword, justly remarks, "The piling of the clinical complexity of rheumatism upon the confusion of allergy at first served only to add to the bewilderment," but he regards the arguments in favour as convincing. Whether one accepts the thesis presented by the author or not, the book is one to be studied by all who are interested in the problem of the rheumatic diseases.

PARODONTAL DISEASE

Parodontal Disease. A Manual of Treatment and Atlas of Pathology. By E. Wilfred Fish, L.D.S., M.B., D.Sc. (Pp. 185; illustrated. 18s.) London: Eyre and Spottiswoode. 1944.

During the last ten years or so a great change has occurred in our attitude towards the group of dental diseases commonly known as pyorrhoea. Too often in the past the dental surgeon and the medical practitioner have advised and indeed demanded the extraction of every tooth in a patient's mouth in order to eradicate a focus of infection. This book describes in detail our present knowledge of the aetiology and treatment of parodontal disease, and shows how with conscientious treatment and hygiene the condition can be cured and the mouth made healthy without subjecting the patient to the distressing ordeal of wholesale extractions. The actual organisms in an uncomplicated case of pyorrhoea are confined to the floor of the minute ulcers on the gum margins and, as the disease progresses, to the ulcers at the bottom of the pockets and not in the deeper tissues. Therefore, the author argues, if the pockets round the teeth can be entirely eliminated, the ulcers allowed to heal, and new, healthy, and hard epithelium be stimulated to grow, the patient is cured if he will undertake by hygiene and home treatment his part in keeping the gum margins hard and healthy.

The early chapters on the aetiology and pathology of pyorrhoea form a sound basis for the development of a system of treatment which is described in detail and contains much original thought, and the aim of which is to promote a firm keratinization of the epithelium of the gum margin by constant friction from the toothbrush and wooden toothpick. The operation of gingivectomy might be described as the "surgery of access" for the wooden toothpick, for until the pockets have been eliminated it is impossible for the patient to carry out his most important routine. There is an excellent chapter on the treatment of acute ulcerative stomatitis which should be of special interest to the medical practitioner; this is directed towards protecting the ulcers by coagulation with chromic acid or with zinc oxide and oil of clove packs. The essence of treatment, in the author's own words, is the constant care and advice and the untiring and patient supervision lavished on every part of the gum margin of every patient from childhood to old age.

Notes on Books

Primer Congreso Nacional sobre Enfermedades Endemo-Epidemicas is a bulky report containing papers read at the Argentine First National Congress on Endemo-epidemic Diseases held in Buenos Aires in 1942. The congress took place under the auspices of the department of pathology and clinical study of infectious diseases of the University of Buenos Aires. The subjects discussed include diphtheria, typhus, brucellosis, scarlet fever, Chagas's disease, hydatid disease, respiratory infections, typhoid fever, and a number of other infectious diseases. It is impossible in the space available to do more than mention the principal topics, but this volume should be of interest to all who are concerned with public health and can read Spanish. Papers by Prof. A. Sordelli and other distinguished Argentine contributors are included.

The Public Attitude towards Mental Disease, by Dr. HORACE HILL, is a short pamphlet privately printed (Laverstock House, Salisbury; 2s. 6d.) with the object of persuading the public to recognize the desirability of the early treatment of mental disease and urging the advantages of life in a mental home for the patient compared to his struggles in the world outside, where he is subject to unfair competition and persistent misunderstanding. The importance of discharge from the home as soon as the patient is well enough is also stressed, and the desirability of encouraging him to revisit the home after his discharge as he would an institution for physical disease is pointed out. In this way stigma would be removed and a more rational attitude towards mental illness fostered. We would all probably agree with the principles enunciated in the pamphlet, and if no scientific presentation or new ideas are expected it may be found useful for distributing to patients' friends.

Col. C. A. WEBSTER'S vade mecum entitled *Regimental M.O.* contains little to criticize and much to commend. By combining brevity with a free and intimate style the author has managed to cover an amazingly wide field. Apart from its obvious value to the newly commissioned, many medical officers with long experience will welcome a handbook of convenient pocket size, reference to which may save wearisome search in A.M.S.Regs. or Standing Orders, R.A.M.C. The booklet is published at 2s. 6d. by A. Wheaton and Co. Ltd., Exeter, and royalties derived from its sale will go to the R.A.M.C. Comforts Guild.

Preparations and Appliances

REDUCTION OF LEG FRACTURES BY HYDRAULIC TIBIA TRACTION

Dr. J. E. STANLEY LEE, F.R.C.S.Ed., medical superintendent, New Cross Hospital, Wolverhampton, writes:

In fractures of the tibia and fibula overriding of the fragments is extremely common owing to sliding in oblique and spiral fractures and loss of apposition in the case of transverse fractures, and considerable difficulty is experienced in correcting the displacement and in maintaining good alignment. Manual reduction is often unsuccessful and, if swelling is marked, redisplacement occurs even despite a well-applied plaster unless continuous traction is also employed. In the new method here described reduction is accomplished by hydraulic tibia traction with the aid of an operating table equipped with an oil-pump base. Though primarily intended for skeletal traction, the method can also be used with strapping extension where displacement is slight or moderate.

Description of Apparatus

The apparatus employed consists of:

(*a*) An operating table equipped with hydraulic raising mechanism (Fig. 1).

(*b*) A thigh support (Figs. 2 and 3) in the form of a half-section of a truncated cone 10 in. long, made of sheet metal suitably padded, having a proximal diameter of 7½ in. and a distal diameter of 6½ in., and attached to a strong base with an angled extension piece which passes round the edge of and below the table, to which it is fixed by a screw clamp operated by means of a sliding tommy bar. At the proximal end the sides are cut away to enable the thigh rest to be used for adolescents and children. Chrome leather pads are fitted to the upper surface of the clamp and to the under aspect of the thigh support. The sliding tommy bar provides good leverage, enables the apparatus to be rapidly fixed in position, cannot become detached, and can be pushed out of the way so as to allow ample room for the surgeon's hand and the passage of plaster bandages. The space available between the edge of the apparatus and the back of the leg is not less than 4½ in. The thigh rest incorporated in Watson-Jones's (1932) tibia traction apparatus, of which the above is a modification, can be used.

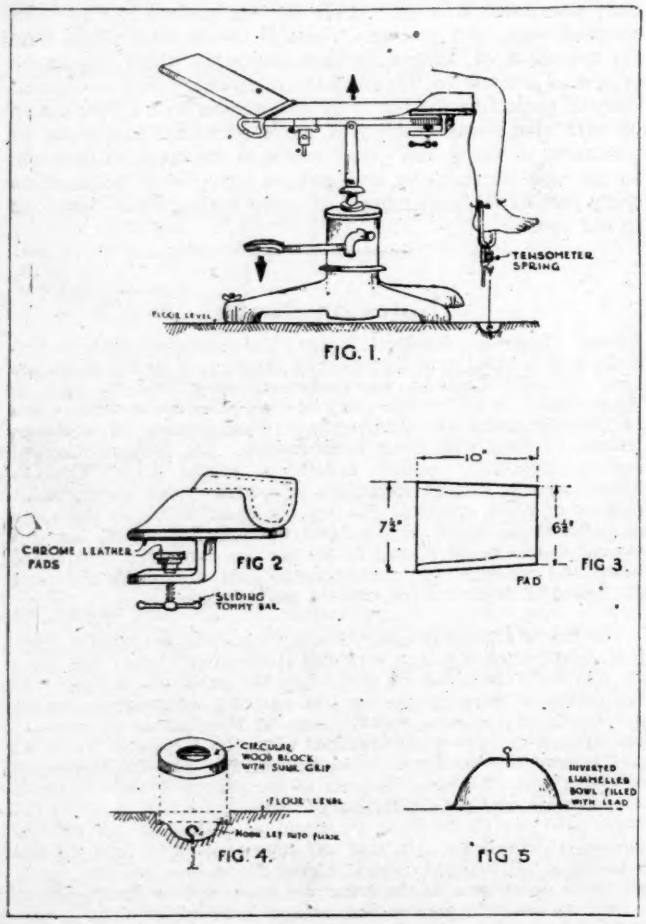


FIG. 1.

FIG 2

FIG 3.

FIG. 4.

FIG 5

(*c*) A tensometer spring (Fig. 1) graduated up to 100 lb., as used by Farquharson (1942).

(*d*) A hook let into the floor (Fig. 4) or, alternatively, an inverted enamelled bowl filled with lead, with a hook inserted through the bottom of the bowl into the lead filling (Fig. 5), which in the case of a bowl of 10 in. diameter weighs approximately 75 lb.

Method of Reduction

A Kirschner wire (or other form of traction pin) is driven through the tibia 1 in. above the ankle-joint, and a stirrup is fixed to the wire (or pin). Next the tensometer spring is hooked to the stirrup and, at its lower end, attached to the hook in the floor (or weight) by a short length of cord (Fig. 1). The thigh is thus supported on the rest with the knee flexed to a right-angle and the limb hanging vertically in the line of gravity, in which position there is no tendency for the fragments to sag backwards, and while the plaster is setting the position of the foot can be controlled by the operator's knee and lateral displacement of the fragments corrected by his hands.

An assistant gently operates the foot pedal attached to the oil-pump base of the operating table while the tensometer spring and cord are held in position until tension is felt. Further elevation of the table by the assistant results in hydraulic traction being applied in the line of gravity. The limb becomes taut, and when the overriding has been fully corrected crepitus is felt by the operator, indicating that the fragments are now in apposition and traction is such that angulation is also corrected. Rotational displacement cannot occur if the axis of the wire is correct, and the toe and the patella consequently point in the same direction. The fragments are then locked against each other by strong lateral pressure of the operator's two hands. This is an essential step, without which accurate reduction cannot be accomplished. So strong is the traction that very firm lateral pressure is necessary. Antero-posterior and lateral radiographs are taken and, if position is satisfactory, plaster is applied.

Application of Plaster

A plaster-of-Paris slab is placed directly to the skin over the posterior aspect of the limb from the back of the knee to the toes passing through the stirrup. A thin pad of wool is placed in front of the ankle-joint; but if swelling has not yet taken place it may be advisable to use a thin wool bandage over the front of the foot and leg before the plaster case is completed by encircling bandages. While the cast is setting, strong lateral pressure is again maintained by the palms of the operator's hands, and as soon as the cast is hard traction is released, the knee straightened to 30 degrees short of full extension, and the plaster case then extended to mid-thigh.

Subsequent Treatment

In all unstable oblique and spiral types of fractures continuous traction is necessary for the first three to six weeks. The limb is supported on a Braun splint, and 10 to 15 lb. of weight suspended over the pulley, with the foot of the bed raised about 12 in. When swelling has subsided a new plaster case should be fitted. The limb is again held in the hydraulic tibia traction apparatus and the new plaster applied, completely unpadded, from the toes to just below the groin with knee-joint slightly flexed. When the plaster is set the Kirschner wire is removed. Radiographs are taken in two planes, and if alignment is not absolutely accurate it should be corrected by wedging the plaster case.

I am deeply indebted to the writings of Mr. R. Watson-Jones (1943), from which I have drawn freely and at length; to the Wolverhampton Borough Surveyor's Department for the illustrations; to Messrs. Guy Motors, Ltd., Wolverhampton, who generously made the thigh support to my design free of charge; and to Mr. McGowan for the weight. Messrs. Down Bros., Ltd., London, have kindly undertaken to manufacture and supply the complete apparatus.

REFERENCES

Farquharson, E. L. (1942). *Illustrations of Surgical Treatment, Instruments and Appliances*, p. 196, E. & S. Livingstone, Edinburgh.

Watson-Jones, R. (1932). *J. Bone Jt. Surg.*, **14**, 591.

—— (1943). *Fractures and Joint Injuries*, **2**, 749, E. & S. Livingstone, Edinburgh.

BRITISH MEDICAL JOURNAL

LONDON

SATURDAY JULY 29 1944

PROBLEMS OF ARIBOFLAVINOSIS

We print in this and last week's *Journal* an abridged form of the Lumleian Lectures of 1944, given by Dr. Hugh Stannus, who took as his subject riboflavin and allied deficiencies. From a survey of the recent literature it would at first seem that the syndrome characterizing riboflavin deficiency was unknown before 1938, when Sebrell and Butler[1] induced the condition experimentally in a group of women in an institution by feeding them on a diet low in riboflavin. They suggested the name ariboflavinosis for riboflavin deficiency, and although they established that lack of riboflavin gives rise to a definite clinical picture they were not the first to describe it. The essential clinical features of riboflavin deficiency were in fact first described by Stannus[2] over thirty years ago, when he was working among the natives of Nyasaland. His observations made in 1911 were published in 1912 and 1913. It is a sad reflection on our colonial administration that these studies should have been made in one of our Colonies in the days of prosperity before the last war; regrettable that little was done to remedy the defective nutrition of these African natives; and disappointing that only recently has the work of Stannus received the recognition it so well deserves. Twenty years before the discovery of riboflavin he described the ariboflavinosis syndrome—the sore tongue and lips, the sodden excoriated palpebral fissures and angles of the mouth (which he termed angular stomatitis), and the characteristic lesions of the anus and external genital organs. He also described certain neurological features and the characteristic smooth red tongue devoid of papillae and epithelium, known later among American workers as the "magenta tongue." While the contemporaries of Stannus believed that these lesions might be infective in character, he thought that they were the result of dietary deficiency. Beyond this he could not go, as the vitamins, though postulated by Funk in 1912, had not then been differentiated and the existence of riboflavin was quite unknown. The importance of this early work of Stannus lay in the fact that he focused attention on the occurrence of this group of symptoms due to a dietary deficiency yet dissociated from pellagra. In the Lumleian Lectures he reviews the work published before that of Sebrell and Butler in 1938. He pays tribute to Fitzgerald Moore, another worker who laboured for many years trying to defeat malnutrition in the Colonies. To Moore belongs the credit of showing that the disease which was rendering thousands of African natives nearly blind was due to a dietary deficiency and amenable to treatment with a yeast extract. In a series of papers between 1930 and 1939 he described certain ophthalmological conditions and nervous symptoms which Stannus thinks may have been due to riboflavin deficiency.[3]

Some doubt has recently been cast upon the validity of angular stomatitis and corneal vascularization as manifestations of riboflavin deficiency. Stannus believes that angular stomatitis is characteristic of the condition, though many workers believe that it is not a specific lesion. Thus it is stated to occur as a result of badly fitting dentures,[4] local trauma, and sensitivity to the constituents of chewing-gum[5] and lipstick.[6] Machella and McDonald[7] failed to improve a series of cases of angular stomatitis by treatment with riboflavin, though some responded to nicotinic acid, vitamin B_6, and yeast. Smith and Martin[8] also observed that angular stomatitis may disappear after the administration of vitamin B_6. Stannus believes that these lesions can be distinguished from the characteristic angular stomatitis of ariboflavinosis, or, as he prefers to call it, hyporiboflavinosis. Even so, a diagnosis of ariboflavinosis should not be made on the existence of stomatitis alone. Considerable discussion has centred around corneal vascularization as a manifestation of ariboflavinosis. Sydenstricker and his co-workers,[9] who drew attention to this sign in 1940, regarded it as pathognomonic of ariboflavinosis and stated that circumcorneal injection, which precedes it, is the earliest and commonest sign of the condition. There appears to have been some subsequent confusion between the terms circumcorneal injection and corneal vascularization. The former is an engorgement of the bulbar conjunctival capillaries of the limbic plexus; the latter is actual invasion of the cornea by capillaries which anastomose to form tiers of capillary loops from which more capillaries develop and extend centripetally. Stannus, who has carried out between 4,000 and 5,000 slit-lamp examinations and is, therefore, qualified to speak on the subject, states that he has seen both circumcorneal injection and corneal vascularization without any evidence of riboflavin deficiency, and that these signs alone are therefore of no diagnostic value in the detection of the deficiency. He agrees that they may occur in patients with riboflavin deficiency, but denies that they are pathognomonic of the condition. His experience confirms that of a number of other investigators who have carried out nutrition surveys during the last two or three years, and have shown that corneal vascularization is fairly common in the population at large in the absence of nutritional deficiency. Sanderstead,[10] for example, found that the incidence of the condition ranged from 80 to 93% in the groups he studied.

A number of workers in the English colonies, including Fitzgerald Moore,[2] Scott,[11] and Landor and Pallister,[12] who all wrote before the recognition of ariboflavinosis as a clinical syndrome, described certain neurological symptoms which did not fit into the clinical picture of beriberi or pellagra. These included burning of the feet, muscular

1 *Publ. Hlth. Rep. Wash.*, 1938, **53**, 2282.
2 *Trans. Soc. trop. Med. Hyg.*, 1912, **5**, 112; 1913, **7**, 32.
3 *West Afr. med. J.*, 1930, **4**, 46; 1932, **6**, 23; 1934, **7**, 119; 1937, **9**, 35; *Lancet*, 1937, **1**, 1225; *J. trop. Med. Parasitol.*, 1939, **42**, 109.
4 *J. Amer. med. Ass.*, 1942, **119**, 790.
5 Ibid., 1941, **116**, 131.
6 *Arch. Derm. Syph.*, Chicago, 1938, **37**, 597.
7 *Amer. J. med. Sci.*, 1942, **203**, 114; 1943, **205**, 214.
8 *Proc. Soc. exp. Biol.*, N.Y., 1940, **43**, 660.
9 *Publ. Hlth. Rep. Wash.*, 1940, **55**, 157; *J. Amer. med. Ass.*, 1940, **114**, 2437.
10 Ibid., 1942, **57**, 1821.
11 *Ann. trop. Med. Parasitol.*, 1918, **12**, 109.
12 *Trans. roy. Soc. trop. Med. Hyg.*, 1935, **19**, 121.

weakness, ataxia, incoordination, paraesthesiae, and loss of visual and auditory acuity. Stannus accepts these symptoms as manifestations of ariboflavinosis, particularly "burning feet," which he believes is a characteristic neurological finding. To these symptoms he adds others which he has observed. They fit into a condition resembling the cerebellar syndrome—muscular asthenia and hypotonia, jerky clonic contractions, nystagmus, tremor, dysdiadokokinesis, a typical gait, and pendulum knee-jerks. It is interesting to note that American workers, who have contributed the bulk of the literature on ariboflavinosis since 1938, do not mention any neurological manifestations. They describe diminished visual acuity, which might be the result of retrobulbar neuritis.

No satisfactory theory has so far been put forward to explain the pathogenesis of the signs and symptoms of riboflavin deficiency and their peculiar localization. Why are the mucocutaneous junctions of the mouth, eyes, and anus selected? Why is the tongue affected? Why do the nervous symptoms resemble the "cerebellar syndrome"? Stannus has advanced a novel theory based on anatomico-physiological considerations to explain these varied lesions. He believes that they are a manifestation of an acute functional derangement of the capillary circulation of the affected parts. The first tissue to suffer from riboflavin deficiency is the endothelium of the capillary system, which needs riboflavin for its nutrition. The capillaries undergo a reversible functional disturbance, a capillary dysergia, resulting in loss of tone and dilatation. Normal cellular metabolism is upset and tissue functions are disturbed. The first parts to suffer are those with the largest numbers of capillaries, those with the greatest metabolism, and those with a very specialized function. The interference with tissue-cell metabolism is probably of the nature of an anoxia, using this term in its widest sense. If the anoxia is not too prolonged recovery of function takes place in the capillaries when supplied with adequate riboflavin; otherwise irreversible processes leading to pathological changes occur. According to Stannus the skin lesions of ariboflavinosis, which occur where the skin is thin or highly specialized, as at the mucocutaneous junctions about body orifices (lips, palpebral fissures, nares, prepuce, vulva, anus, scrotum), are due to capillary congestion, with resulting impaired nutrition of the skin. The colour of the "magenta tongue" of ariboflavinosis is the result of capillary dilatation and a sluggish blood flow. While Stannus grants that the changes in the cornea are due to riboflavin deficiency, he believes that the visual disturbances associated with the latter have no immediate connexion with the eye but are part of a lesion in the central nervous system. The neurological manifestations, says Stannus, are the expression of a metabolic disturbance in nerve tissue produced by capillary dysergia, the more vascular tissue being the first to suffer—i.e., the grey matter and cerebellar neurophil.

Any satisfactory theory of the pathogenesis of ariboflavinosis, particularly one involving the capillaries, must be based on histopathological findings. From the pathologist's point of view, if not from the clinician's, it is disappointing that no cases of human riboflavin deficiency have come to necropsy so far. Studies with the dermal capillaroscope in the living might help to support the theory of Stannus. It has been shown with the use of this instrument that there are capillary changes in the skin in pellagra.[13] A microscopical study of the skin of subjects with riboflavin deficiency might yield interesting results.

THE "TRIPLE" AND THE "CONJOINT"

If effect is given to the recommendation of the Goodenough Committee that every medical school should be an integral part of a university, one school in London and three in Scotland will either cease to teach or undergo absorption. The West London Hospital Medical School is the latest in the field of undergraduate medical education; it was opened in 1937, and in the session 1942–3 it had 38 students, all women. The decision on its application for admission to the University of London was deferred pending a general review of the provision for women students. The Goodenough Committee is of opinion that an increase in the number of University of London schools cannot be justified, and that, important as the contribution of this school to the training of women students has been, it should not continue its undergraduate work beyond possibly the next four or five years.

The three Scottish schools have a different background. The Anderson College of Medicine in Glasgow was established as part of "Anderson's University" in 1795. John Anderson, professor of natural philosophy and friend of James Watt, endowed the school with a meagre legacy, out of keeping with his rather grandiloquent prediction for it; but it struggled on and prospered, and came to have more teachers than the University of Glasgow. Later its medical side was separated from the other departments of technology and given its own domicile and governing body. The second school, St. Mungo's College, Glasgow, owes its origin to the steps taken to rehabilitate the Royal Infirmary after the severe blow it received when the University was removed to the west side of the city and the Western Infirmary built alongside. The Royal Infirmary School of Medicine, for which a charter was obtained in 1889, became St. Mungo's College. The third school, that of the Royal Colleges of Edinburgh, consists of lecturers licensed by the two Colleges and in most cases recognized by the University. It is a single corporate body governed by a board. In the last academic year for which figures are available Anderson College had 252 undergraduate students, St. Mungo's 339, and the School of Medicine of the Edinburgh Royal Colleges 359. From these three schools the majority of the candidates for the Scottish triple diploma are drawn.

Extramural teaching—there were other schools of the kind which flourished in Glasgow for a short time in earlier days—has played a great part in Scottish medical education. The rostrums of these schools attracted teachers of eminence, especially the great anatomists. The schools were able to parallel the courses in the University; their fees were lower, and they offered opportunities for teacher and taught which the more rigid methods of the University

[13] *Acta Neurol. Psiquiat.*, 1940, 1, 198.

denied. But university teaching itself altered and widened. A large increase took place in the number not only of professors but of departmental lecturers and assistants, and facilities were offered for research, so that the abler of the young medical graduates tended to seek appointments in the University rather than in the extramural schools. The tendency is all towards integration with the University, with its liberal atmosphere and intellectual stimulus and the contacts it offers with other learning. If in this process extramural teaching disappears it is an evolutionary development and takes nothing from the lustre of the past achievement of these schools.

In another direction the Goodenough Committee suggests further concentration upon the university. In urging that the University of London—and this also applies in a measure to Oxford and Cambridge—should reconsider the standard of its final examination, introducing "internal" features, bringing it more into line with its teaching, and holding the examination more frequently, also that university grants to schools might be conditioned by the number of students taking the university degree, the Committee is obviously placing the English Conjoint Board examination in some peril. The history of the "Conjoint" goes back to the latter part of the nineteenth century. In 1873 we find the Medical Reform Committee of the British Medical Association expressing satisfaction with steps taken to form a Conjoint Examining Board for England, but it was not until ten years later that the two Royal Colleges agreed that a diploma be granted in conjunction on the results of the final examinations held on their behalf by the Examining Board. Thereafter the licence of the Royal College of Physicians and the diploma of membership of the Royal College of Surgeons were no longer granted separately as qualifying for admission to the *Register*. The conjoint examinations in England and also those in Scotland (by the Colleges of Physicians and Surgeons of Edinburgh and the Faculty of Physicians and Surgeons of Glasgow) were approved by the General Medical Council in 1884. The Medical Act, 1886, laid it down that a qualifying examination might be held for the purpose of granting a diploma conferring the right of registration by "any combination of two or more corporations in the same part of the United Kingdom who may agree to hold a joint examination in medicine, surgery, and midwifery, and of whom one at least is capable of granting such diploma as aforesaid in respect of medicine and one at least is capable of granting such diploma in respect of surgery." The Conjoint Board examinations in England have proved very popular, especially among London students, owing to the attractive alternative they offered to the London University Final M.B., B.S., which is out of line with the teaching of the school, and is a stiff "external" examination, reminiscent of the days when the university was an examining body only. The "Conjoint" enables Oxford and Cambridge students to qualify a year earlier than they can do by taking their university degrees. Of the practitioners who qualified from London schools in 1933–7, 2,414 were registered on their diploma of the English Conjoint Board, and only 192 on a university degree, though it should be added that a university degree was obtained later by about one-third of them. From the other universities 414 were registered on the Conjoint diploma.

One incidental result of a decline in popularity of the Conjoint examination would be a diminished revenue of the Royal Colleges. The amount which the Royal College of Surgeons received from the Conjoint Examination Board in 1942–3 was £11,168—more than half the total income of the College. But it is not to be disputed that competition between the university and the Conjoint Board examinations is educationally bad, and that a university examination ought to be regarded, not as something separate from the student's record during his course of training but rather as a final check upon it. If, again, as in the case of the extramural schools, the portended changes in medical training threaten the numerical pre-eminence of the "Conjoint," no reflection will be conveyed on the diploma, for it is agreed that the examination is of good vocational standard, only slightly different from that of the university, and with the same subjects.

SURGICAL TREATMENT OF CORONARY DISEASE

It is now some seven years since surgeons first attempted to provide a new blood supply for the heart which was being starved by progressive coronary sclerosis. Claude Beck was a pioneer in this field of surgery in America, and in two recent papers he and his physician colleague Harold Feil review the results of the past few years.[1 2] It appears that there have been few new discoveries or startling improvements in technique. Some might have come from this country had it not been for the early death of Laurence O'Shaughnessy, whose operation of cardio-omentopexy had already given promising results.

During his experimental work on dogs Beck observed that the gradual reduction of the blood supply to a small area of the heart, by ligation of the arterial branches supplying the area, results in a "trigger" mechanism. When a certain degree of anoxaemia has been reached further slight reduction in the blood supply causes sudden ventricular fibrillation and death. In contrast two of the main coronary arteries may be partially occluded, producing a less degree of anoxaemia over a larger area, without causing death. Beck draws two conclusions from these observations: first, that acute anoxaemia of a small part of the cardiac muscle can act as a trigger or irritable zone which may throw the whole mechanism into the convulsions of ventricular fibrillation; and, second, that a very small improvement of the blood supply to the irritable zone can reduce its irritability and prevent it from "firing." The blood supply to the heart can be increased by effecting communications between the coronary vessels and other arteries outside the heart—for instance, by grafts, or by producing intercoronary anastomoses. The coronary arteries communicate very little with one another, but inflammation on the surface of the heart produces anastomotic channels between them. Beck showed that ligation of the descending ramus of the left coronary artery in normal dogs caused death in 70% of cases, but when the surface of the heart had previously been abraded to set up inflammation and adhesions the mortality after ligation of this vessel was only 38%. Beck's operation aims at producing inflammatory adhesions between the heart and

[1] *Ann. Surg.*, 1943, **118**, 788.
[2] Ibid., p. 807.

the parietes by applying powdered asbestos to the surface of the heart and by laying grafts of mediastinal fat against it. His illustrations demonstrate conclusively that vascular channels are formed in such adhesions, both in animals and in man.

Unfortunately the results of this procedure are not encouraging. Feil describes 37 patients treated by the Beck operation: there were 14 operative deaths, and 23 survived. Of the latter, 14 were improved and are still living, 9 died within four months to six years of operation, all from coronary disease, and 5 of these experienced relief of their symptoms. Patients with severe coronary disease rarely survived the operation, and those that did survive were unimproved. Therefore the early case with only slight myocardial damage should be chosen for operation; but even in the later cases of the series, when such selection was being applied, the operative mortality was 35%. Grave as is the prognosis of coronary disease, such a mortality will discourage most physicians from submitting their patients to this hazard. Nevertheless there is now considerable evidence that these operations do, in most cases, reduce the severity of the symptoms and even prolong life if the patient can survive the initial shock. It is therefore to be hoped that future improvements in technique will lower the operative mortality. There is certainly scope for more research in this field.

HEALTH RECORD OF THE U.S. ARMY

The Surgeon-General's statistical report for 1943, circulated by the U.S. Office of War Information, gives some interesting facts and figures. The number of men examined during the year was 5,200,000; the number recruited into the Army was 2,400,000, and into the Navy, Marine Corps, and Coast Guard 900,000; the number rejected was 1,900,000 (36%). The Army certificate-of-disability-for-discharge rates have run rather high. The majority of such discharges occurred within the first three months of service, suggesting that mental and physical defects were missed at the medical recruitment examinations. The health of the U.S. Army has been better than it was at any time during peace. Up to the end of 1943 only 3% of the wounded had died of their wounds. The Army's over-all death rate from disease was 0.6 per thousand per annum, this despite the fact that American soldiers are fighting on all fronts, in all climates, and exposed to all diseases. The death rate for pneumonia before the advent of the sulphonamide drugs was 24%; the present Army rate is 0.7%. The death rate from meningitis, which forty years ago killed 80% of those contracting it, was reduced to 38% in the last war and, through the use of "sulpha" drugs, has now fallen to 4.2%. The admission rate for tuberculosis has declined from 12 per thousand per annum in the last war to 1.2 in the present war. The programme for recruitment of individuals with venereal disease was officially inaugurated in October, 1942. Special V.D. hospitals were built at each of the 34 reception centres, with a total of 6,510 beds. Recruits having venereal disease were referred to these hospitals, where they were examined and treated. The U.S. Army is vaccinated against smallpox, paratyphoid, typhoid, and tetanus. Typhoid fever, which was the scourge of armies until 1912, has been reduced to insignificance. There have been no deaths from typhus, though this disease is endemic in North Africa. There has been no tetanus in the vaccinated, and vaccination against yellow fever has prevented any incidence of this disease. Vaccination is available when there is danger of exposure to cholera and plague. Against the menace of insects as carriers of disease the sanitary service has developed the Freon pyrethrum bomb which, upon the turning of a spigot, will spray a plane or tent or barrack killing all flies, mosquitoes, and bed-bugs in the area treated. This spray is non-toxic to man. The synthetic compound known as "DDT" (dichlordiphenyltrichlorethane) has been introduced for killing lice; the use of this powder (which is also effective against flies) enabled the United States Typhus Commission to prevent the development of an epidemic of typhus fever in Naples. The report on the dental service states that rejections for dental defects ran to around 5%. It was decided then that if a recruit had no malignant disease or extensive osteomyelitis in his jaws he would be accepted regardless of the number of serviceable teeth. This threw an immense load upon the Army Dental Corps. During the years 1942 and 1943 14,600,000 cases were treated, and 31,000,000 teeth were filled; more than 1,400,000 bridges and dentures were supplied, 196,000 dentures mended, and more than 6,000,000 teeth replaced by dentures and bridges.

MEDICAL FILMS

There should be plenty of room for the use of the moving film in medical education. If anyone had doubts on this subject the viewing of the two films recently made by the British Council should have removed them. The moving picture tells a story that often enough can be told with poor effect in words only. In fact there is always the risk that the word will accumulate many meanings remote from what it stands for. One such word is "rehabilitation," which at the moment is in danger of almost becoming a political slogan. The recent British Council film, "Accident Service" (see *B.M.J.*, June 10, p. 789), made it vividly plain to all and sundry just what rehabilitation is. We can think of no better way of informing the medical student of what rehabilitation is than by showing him this film. It is even possible that the film will teach more than a visit to such a centre as Berry Hill Hall, because the film is the product of many minds, and gathers up into one whole all the numerous links in the medical and surgical chain that rehabilitates the injured or diseased person. But films can become an educational force in medicine only if they are well done, and they can be well done only if the expert film technician and the expert medical technician are given a free hand to work out their common problem on an equal footing. The film technique should be as good of its kind as the medical or surgical technique if the student's mind is to be satisfied. Until the perfect library of medical films can be formed we must rely upon the slightly imperfect copies that owe their existence usually to the enthusiasm of amateur medical cinematographers. Full use of the films that now exist can be made only if they are fully catalogued and classified by some central organization. Recently the Scientific Film Association set up a medical standing committee "to further the production and distribution of medical films suitable for circulation within the medical profession, either for training and teaching purposes or for the general dissemination of information." After the war it is hoped to set up a central medical film library, and as a step towards this any doctors who know of or have medical films are asked to inform the secretary of the Medical Standing Committee, Dr. S. J. Reynolds, 14, Hopton Road, Streatham, S.W.16. So that the information about each film may be systematically assembled a questionary has been drawn up by the Scientific Film Association which will be sent to any inquirer. Ordinary membership of the Scientific Film Association is open to anyone interested in the subject.

SOME COMMON PAEDIATRIC PROCEDURES

A REASSESSMENT FOR CHILD WELFARE PRACTICE*

BY

DOYNE BELL, D.M.

Physician, Belgrave Hospital for Children; Physician in Charge of Children's Department, Queen Mary's Hospital for the East End

It has seemed to me suitable, in addressing this audience, to confine my remarks to such aspects of the subject as are the day-to-day concern of the worker in the child welfare clinic. The choice of a few points of common paediatric procedure for your reconsideration has therefore been based on cases referred to me from welfare centre practice or presented as problems to midwives and students in maternity departments. If, then, my conclusions seem to many to be self-evident, I must plead that to others they have seemed less obvious.

The Crying Baby

Handbooks on the care of babies quite rightly tell their readers that the treatment of the crying baby is to find out the cause of its crying and correct it; yet in practice it is not at all uncommon for mothers to complain that their babies cry for hours on end, and an examination of such babies shows no abnormality to account for this behaviour. Endless and apparently motiveless crying is extremely disturbing to the mother, and may result in such anxiety that lactation fails. Even without such a serious consequence the symptom is distressing both to the baby and to all who hear it, and calls for treatment. I think in many cases, though the crying was started by some momentary discomfort, its persistence is due to the discomfort occasioned by the crying itself. The baby gets hot and its pillow wet, and so the crying continues. In these cases, after excluding by thorough examination an organic cause of pain, the giving of a sedative is amply justified. It is important, however, to give it in sufficient dose to ensure that the habit of crying is broken. Nothing discourages the mother more than to be given a medicine which she hopes will soothe the infant and then to have her hopes frustrated. For an infant 6 months old, for instance, phenobarbitone, gr. 1/2, would be an appropriate dose. The sedative need not be repeated for more than three or four nights as a rule.

Breast-feeding

The commonest errors in the handling of the breast-feeding of the newborn child seem to be due to anxiety and pessimism—anxiety lest the newborn infant should be underfed in the early days of life, and pessimism with regard to the mother's ability to suckle her child. Admittedly the factors influencing lactation are incompletely understood and complex—too complex for detailed discussion here; but it is clear that successful breast-feeding does not depend entirely on mechanical factors such as the stimulation of the nipple by sucking and the emptying of the breast. Endocrine influences of much complexity play an important part, and anxiety or pessimism or a display of even cheerful ignorance by nurse, doctor, or health visitor will often effectively disturb what should be a normal lactation. My plea, therefore, is for a much more intelligent and intensive field study of lactation. Such study would show, among many things, how great is the variation in the appetites of normal newborn babies and in the time taken for lactation to become established. I am depressed at the number of infants discharged from maternity hospitals at the age of 2 weeks or less, wholly artificially fed. Often in such cases I learn that on or about the tenth day after delivery the mother has been given tablets to dry up her milk and told that she has an insufficient supply. There is nothing pathological in the fact that on the tenth day the supply of breast milk is less than 2½ fl. oz. per lb. of the infant's body weight. I think it is a fair generalization to say that, in the case of a healthy mother and infant, it is never possible in the first three weeks to state that lactation will not be established and should be abandoned. I have made it a rule, in a midwifery department where the newborn babies are in my care, that no infant is ever to be given a complementary feed (I except water and saline) without my being notified. As a result such feeding is, in normal cases, practically unknown.

In contrast let me instance a case referred to me last year. A mother aged 20 was sent with her 9-weeks-old infant to see me because he was restless and crying, and vomited. The baby had been born in a private nursing home, and had never been put to the breast. The mother had been purged and had her breasts bound up on the third day, and a variety of proprietary foods had been given to the infant. On examining the mother's breasts I found that a few beads of milk could be expressed. She was obviously disappointed that she had been thought incapable of suckling her child. I gave her the ordinary advice for re-establishing lactation, but warned her not to be unduly depressed if she failed. She wrote 4½ weeks later to tell me that her infant was fully breast-fed. The indiscriminate use of synthetic oestrogens has of course made the re-establishment of wrongly abandoned lactation more difficult.

The study of natural lactation can be carried out only by the paediatrician who sees the baby from birth onwards. The time available to the obstetrician or midwife—two or three weeks as a rule—is too short for a proper study to be made. This, incidentally, is a further argument, if one is still needed, for the paediatric supervision of the newborn babies in maternity units. The premature termination of lactation, like the termination of pregnancy, is a step which may have serious consequences, and should never be undertaken without a full knowledge of the factors involved and a careful consideration of the medical indications.

The formation of an abscess in the lactating breast is a complication of breast-feeding that needs much study. There is apparently a widespread failure to appreciate the aetiological factors and what should be the aim of treatment. Most cases of suppuration in the lactating breast are preceded by obstruction of the milk ducts, producing a tender, knotty breast. Treatment of this condition and prophylaxis of abscess formation should clearly aim at removing the obstruction and re-establishing the milk flow. Taking the baby off the affected breast and tight bandaging are therefore to be condemned.

When an abscess has definitely formed it is still too often forgotten by the surgeon that he has two patients—the mother and the infant. No treatment of breast abscess should be considered entirely satisfactory which does not achieve as its end-result normal lactation. A fissure in the nipple should be considered a danger signal, for it may be a portal of entry for infection to the deeper breast tissues. In any event, the fissure itself is extremely painful and may render breast-feeding from the affected side temporarily impossible. If, on account of pain, breast-feeding has to be suspended, particular care must be taken, while the fissure is under treatment, to prevent milky engorgement of the breast, either by manual expression or by the use of the breast pump. The objects of local treatment of the fissure are disinfection and rest, just as if it were an ulcer anywhere else in the body. There are many ways of achieving these ends, but a very practical method, and one that gives rapid relief of pain, is the application of a lead nipple-shield between feeds.

Artificial Feeding

The technique of artificial feeding should be elastic and be modified to suit the individual infant's requirements, and in this important branch of paediatrics rule-of-thumb methods have little place. One not uncommonly sees infants suffering from dietetic upsets who have been treated by many changes of the brand of dried milk offered but none in the composition or quantity of the food. The only change has been in the branded name on the tin. The slavish belief in the advertised merits of proprietary brands of dried milk has interfered with the wider use of national dried milk. In this respect some hospitals have been much to blame. An infant is admitted to hospital and fed on a proprietary brand of straight full-cream dried milk; if he does well the mother is naturally reluctant to change to national dried milk when the infant returns home. Thus she pays at the chemist's shop three or four times as much as she would pay for the identical product at the food office or clinic.

Respiratory Infections in Childhood

Chronic nasal discharge in small infants with resulting snuffles or nasal obstruction is a very common symptom. The origin of these snuffles is probably a mild catarrhal infection of the nose, but the persistence of the symptom is often due to mistaken attempts to clear the nose with pledgets of cotton-wool. These pledgets are useless for the purpose for which they are intended, and the delicate mucous membrane of the infant's nose is always irritated and often injured by their use, and a chronic infection results. Most cases of snuffles will clear up when the use of such pledgets is discontinued.

Whether tonsils should be removed or not is a question asked frequently in welfare centre practice. It clearly requires careful and individual consideration. No one who has seen some of the bad results of indiscriminate tonsillectomy will fail to realize the importance of a right decision. The tonsil undoubtedly plays a large part in the defence of the respiratory tract of the young child against infection, and its removal while still capable of healthy function will only weaken the defence. I do not think it is an over-simplification of the problem to state that there are but two indications for the removal of the tonsils in children under 5 years old. The first is hypertrophy of the tonsil to a degree which interferes with the child's swallowing; the second is chronic infection of the tonsil, resulting either in repeated auto-infection of the respiratory tract or in the child's becoming a carrier of pathogenic organisms which cannot be eliminated by other means. It is important to remember that a history of repeated acute infections in the child is not necessarily an indication for removal of the tonsils. The repeated infections may be due simply to repeated exposure to an outside source of infection. The same principles

* Lecture (abridged) given in London to the Maternity and Child Welfare Group of the Society of Medical Officers of Health.

should be applied to consideration of the removal of adenoids. The adenoids should be removed if they are the site of chronic infection, or if their hypertrophy obstructs the nasal air-way or interferes with the drainage of the paranasal sinuses or middle ear.

In the prophylaxis of respiratory infection in childhood it is still not uncommon to find that clinics are exposing children to ultra-violet light. The uselessness of this procedure was well shown by Dora Colebrook as long ago as 1929. She studied three groups of children, of which one was exposed to ultra-violet light, another to a screened lamp from which no ultra-violet rays came, and the third served as a control. She found no significant difference in the incidence or duration of respiratory infections in the three groups; the very small and insignificant difference being favourable to the control group. In the absence of any work to contradict these findings it seems to me that the exposure of children to ultra-violet light is quite unwarranted.

Strawberry Naevi

The strawberry naevus or capillary haemangioma is quite a common birthmark, and a great number of cases are still referred to hospital, often with the suggestion that they should be treated with CO_2 snow, with radium, or by excision. The natural history of these naevi was studied by W. A. Lister (1938), and he found, in a series of 77 children with 93 naevi who were watched for periods varying from one to seven years, that there was no exception to the rule that naevi which grew rapidly during the early months of life subsequently retrogressed and, of their own accord, disappeared, on the average at about the fifth year.

My experience is in accordance with Lister's findings. Any form of treatment is bound to leave a scar, whereas natural retrogression of the naevus leaves normal healthy skin. The chief difficulty in practice is to persuade the child's mother that the naevus will retrogress after reaching its maximum size at the age of about 12 months. My practice is to make an exact tracing of the naevus on a piece of "cellophane" or washed *x*-ray film and transfer this pattern to paper. This paper is dated and given to the mother to keep. She may then observe for herself that the naevus does not further increase in size, but gradually shrinks or fades. In many cases the retrogression starts in patches in the middle of the naevus, but after the age of about 12 months the edges, even if they are the last bits to disappear, do not spread.

Circumcision

This mutilation has a long history which is of great anthropological interest. But its popular justification on hygienic grounds seems to be of quite recent origin and of extremely doubtful validity. As it is not free from hazard and discomfort it is well, when considering it as a surgical procedure, to take stock of the indications for the operation. So far as I know, there is but one—phimosis; this being defined as a stenosis of the preputial orifice which prevents retraction of the prepuce behind the corona of the glans penis. In most cases in which circumcision is performed no attempt is made to assess whether phimosis is present or not; for normally the prepuce of the infant is lightly adherent to the glans, and it is these adhesions which prevent retraction. If the adhesions are divided—and this can easily be done either by digital retraction or with a probe, though I do not suggest that it is a desirable routine procedure—it will be found that phimosis is rare. If the preputial orifice is smaller than the urinary meatus—an extremely rare occurrence—interference with urination will occur and operation is certainly necessary. But I doubt whether operation is indicated in lesser degrees of phimosis in early infancy. Indeed, it has been suggested that some degree of phimosis, as above defined, is normal in the young infant.

I have heard it suggested that a long and inflamed prepuce or the so-called "cauliflower prepuce" indicates circumcision. This condition is in fact an absolute contraindication to circumcision. The "cauliflower prepuce"—the site of cellulitis, oedema, and even ulceration—is the result of an ammonia dermatitis. To remove such a prepuce is to expose the more delicate glans and the urinary meatus to the same trauma with a far more painful result—namely, meatal ulceration. One might as well urge a man to remove his steel helmet in an air raid because it was being dented by falling "flak."

REFERENCES

Colebrook, Dora (1929). *Irradiation and Health*, Med. Res. Cncl. Sp. Rep. Ser. No. 131, London.

Lister, W. A. (1938). *Lancet*, **1**, 1429.

P. A. O'Leary and W. E. Herrell (*Proc. Mayo Clin.*, 1944, **19**, 20) record the treatment of a case of late cutaneous syphilis with penicillin. The patient was a 42-year-old married woman who for eight years had had a nodular syphiloderm of the nose. She was given 20,000 units of penicillin intravenously twice a day for 8 days. The result is described as striking, and three weeks after the course of treatment all that was left was some residual pigmentation. Serum tests for syphilis remained unchanged.

THE LISTER INSTITUTE

Long-term Investigations

The report of the Governing Body of the Lister Institute of Preventive Medicine, given at the recent annual meeting, contains brief accounts of many continuing investigations which cannot be expected to reach finality in any one year. Some of the members of the staff are on active service abroad or seconded to a Government Department. A number of the workers are enjoying research facilities at Oxford, and others at Cambridge. One event of the year was the establishment by the Medical Research Council, which is administering the London blood supply depots on behalf of the Ministry of Health, of a unit at the Institute for research into, and filtration of, blood plasma and serum for transfusion, and the Director of the Institute, Dr. Alan N. Drury, is chairman of the Blood Transfusion Research Committee.

Two of the workers, Dr. R. A. Kekwick and Dr. M. E. Mackay, have been studying problems arising in the large-scale processing of human serum and plasma. They have found that the adjustment of the reaction of plasma to *p*H 5.4 with sterile citric acid before ether extraction provides a method for the quantitative removal of fibrinogen, as well as for removing the unstable lipoid complexes. After readjustment of the product to *p*H 7 followed by Seitz filtration, a transfusion fluid is obtained which remains stable for at least twelve months. It is added that problems arising in the kaolin processing of plasma have been surmounted and the Serum Unit has been filtering the entire output of the London blood transfusion depots before dispatch to Cambridge for freeze-drying.

Serological and Bacteriological Studies

One of the serological studies undertaken concerns the treatment of louse-borne typhus by anti-proteus OX 19 serum. Dr. A. Felix has been working on this serum from the horse, and a few preliminary trials have been carried out in North Africa in collaboration with Capt. T. E. Woodward of the United States Typhus Commission. Six selected severe cases were treated; three patients received the serum intravenously and three intramuscularly. All did well, though one of them had been in a comatose state for several days. These trials have shown that large doses of the serum are well tolerated, and that as a result of the injections an initial low-titre OX 19 reaction in the patient's serum can be increased from twenty to thirty times.

Dr. Felix and Miss Callow have continued the typing of typhoid, paratyphoid, and food-poisoning bacilli with the Vi bacteriophage. A paper by them on the typing of paratyphoid B bacilli in this way was published in the *British Medical Journal* of July 31, 1943 (p. 127). One hitherto unknown Vi-phage type of the typhoid bacillus was identified during the past year as being responsible for a small outbreak of typhoid fever. This type does not seem to be indigenous to the British Isles, as it has been traced to a chronic carrier who acquired the infection during the war in South Africa more than forty years ago. On the other hand, a Vi-phage type of *Bact. paratyphosum* B has been found which must be considered indigenous. The number of known Vi-types and subtypes of the paratyphoid B bacillus is now five.

Nutritional Studies

The Ministry of Food has been anxious to ascertain more accurately the requirement for vitamin A as derived from preformed vitamin A and from the provitamin carotene. This has been done by a human experiment carried out on volunteers, who were conscientious objectors, at the Sorby Institute at Sheffield. Since July, 1942, these volunteers have received a diet made as deficient as possible in vitamin A and carotene; a small number have served as positive controls receiving supplements of these substances. Observation was specially directed to the measurement of capacity for dark-adaptation. At the end of a year none of the volunteers showed any definite signs of deterioration of the visual threshold, but after fourteen months one subject began to show this deterioration, with slight skin symptoms, and a very low value for plasma vitamin A, all of which signs progressed rapidly until he was given a small daily dose of the vitamin, when the symptoms slowly regressed. The experiment has indicated how large are the vitamin A reserves in the liver of the healthy human adult,

and that a daily dose of vitamin A, smaller than that usually recognized as the daily requirement, is enough to stop depletion and restore the organism to health, though whether to perfect health is still uncertain.

The nutritive value of the nitrogenous substances of the potato has been under investigation. Weight for weight, the nitrogen of the potato has been found to have a biological value at least equal to that of the proteins of whole wheat, though only about one-half of the potato nitrogen is in the form of protein. It is believed that a supplementary action occurs between the protein and non-protein nitrogenous substances in the tuber, probably attributable to the amino-acids present in the latter, which explains why the total nitrogen of the potato, though half is non-protein, has at least as high a nutritive value for growth as an equivalent amount given in the form of potato protein. The nutritive value of proteins from other foods has been found to be enhanced by the addition of protein-free potato juice.

A nutritional survey of women employed in Oxford factories and of local housewives of similar age and social position was carried out from the autumn of 1942 to the end of 1943. On the whole the nutritional state deteriorated very slightly during that period, but more so among the factory workers than among the housewives.

Other Researches

Work on the cultivation of vaccinia virus on the chorio-allantoic membrane of the developing chick has been resumed with a view to preparing sufficient quantities of suitable virus for field trials and for clinical use in vaccination. The differences in the lesions inflicted on this membrane by strains of virus derived from sheep and those derived from the calf, which were reported four years ago, are being made the subject of further investigation, and it is hoped to determine which strain is the more suitable for Jennerian prophylaxis. Observations are also being made to determine the best method of preserving stocks of cultivated virus and a convenient form in which it can be issued for clinical use.

A large number of studies are recorded on the effects of different combinations of hormones and vitamins on young adult and old rats. Thus vitamin A, B, or C, administered alone, caused only slight changes in the liver and kidneys, but striking changes followed administration of thyroid hormone in non-toxic doses, including pronounced hyperplasia of hepatic cells, hypertrophy of kidney tubules, etc. Simultaneous administration of the vitamins with the hormone did not significantly alter the effect of the latter. Large doses of the hormone produced a pronounced atrophy of cells and other changes, and here vitamin B had a beneficial effect (regenerative hyperplasia of cells).

Many other biochemical, biophysical, and physico-chemical experiments are recorded.

ORDER OF ST. JOHN OF JERUSALEM

The following medical promotions in and appointments to the Venerable Order of the Hospital of St. John of Jerusalem have been announced:

As Knights, Capt. H. F. Powell, M.B.E., Lieut.-Gen. Sir Alexander Hood, K.C.B., C.B.E., K.H.P., Lieut.-Gen. Sir Gordon G. Jolly, K.C.I.E., I.M.S., Drs. F. C. Bottomley, O.B.E., and W. Stewart. As Commanders (Brothers), Drs. C. Gardiner-Hill, K. S. Maurice-Smith, F. L. Newton, and C. E. C. Wilson, Brig. W. W. S. Johnston, C.B.E., D.S.O., *M.C.*, Major-Gen. P. S. Mills, C.I.E., I.M.S., Col. H. H. E. Russell, O.B.E., V.D. As Associate Commander (Brother), Dr. A. D. E. Behram. As Officers (Brothers), Drs. H. J. O'D. Burke-Gaffney, O.B.E., H. C. Sinderson, C.M.G., M.V.O., O.B.E., S. J. W. Donald, E. A. H. Russell, O.B.E., V.D., H. E. Skeete, O.B.E., J. S. Harbinson, S. F. Chellappah, O.B.E., and N. Caine, Brig. Sir W. S. Duke-Elder, Cols. A. H. Harty, C.I.E., I.M.S., and R. Hay, C.I.E., I.M.S.. Lieut.-Col. W. George, *M.C.*, T.D., Lieut.-Cols. R. F. D. MacGregor, C.I.E., *M.C.*, I.M.S. (ret.), G. Verghese, C.I.E., I.M.S., and J. E. Gray, I.M.S., Capts. T. C. A. Sweetnam and F. W. P. Sullivan. As Associate Officers (Brothers), Lieut.-Col. A. N. Chopra, I.M.S., Major S. A. Paymaster, I.M.S., Capt. G. G. Limaye, Dr. M. N. Mahadevan. As Officer (Sister), Dr. Cornelia B. S. Elgood, C.B.E. As Serving Brothers, Surg. Lieut. Cmdr. R. F. Matters, R.A.N., Lieut.-Col. A. I. Cox, I.M.S., Majors J. D. Murdoch, I.M.S., and A. B. Slack, *M.C.*, Drs. J. H. Pottinger, G. A. Fisher, *M.C.*, T.D., C. R. White, R. St. J. Kemm, W. W. King-Brown, H. C. Campbell, L. A. Watson, H. S. Taylor-Young, R. Brown, A. K. MacRae, K. D. Bean, E. M. McAlpine, C. D. Hough, V. F. Ryan, T. J. Coakley, G. R. Lipp, *M.C.*, K. G. Salmon, P. H. Stewart, S. G. Budd, R. McL. Carmichael, J. A. Davies, and C. E. R. Norman. As Associate Serving Brothers, Lieut.-Col. R. M. Kharegat, I.M.S., Capt. B. S. Chalam, Drs. B. Bose, H. N. Dewan, M. F. Bisni, I. Lipsedge, S. Bloom, P. R. Kappuswami, and P. Vaidyanathan. As Serving Sisters, Drs. Muriel R. Powell, Daphne W. Dear, Margaret Swete, and Alice M. Headwards.

Correspondence

B.M.R. Determinations on Ambulatory Patients

SIR,—I read with very great interest the paper on the determination of basal metabolism on out-patients by Dr. J. Douglas Robertson (May 6, p. 617). The problem of ambulatory basal metabolic rate determinations arose in the Montreal General Hospital approximately 20 years ago. The method investigated corresponded almost exactly with that of Dr. Robertson, and the conclusions were the same. During the last 20 years we have performed approximately 50,000 basal metabolic rate tests on ambulatory patients, and, as Dr. Robertson has pointed out, "these so-called ambulatory determinations are not significantly different from those tests carried out at the bedside under strictly basal conditions.—I am, etc.,

I. M. RABINOWITCH,
Director, Department of Metabolism, Montreal General Hospital.

Why Tie the Cord?

SIR,—I have read with much interest the letters by Dr. E. W. Price (June 3, p. 772) and Dr. K. J. Franklin (June 24, p. 857) on "Why Tie the Cord?" Whether or not it is necessary to tie the cord depends on the presence or absence of a closing mechanism in its vessels. Certain radiological observations focused our attention on the intra-abdominal portion of the umbilical arteries. For the past year I have been examining sections of these arteries. A full account of the results will be published elsewhere, but the following notes, some of which are based on this work, may be of interest with regard to the present question.

In the intra-abdominal portion of the umbilical arteries sub-endothelial ridges are found which project into the lumen of the vessel. These ridges lie within the internal elasic lamina and the tunica media. They consist mainly of longitudinal and oblique muscle fibres (see Fig. 1). Their prominence

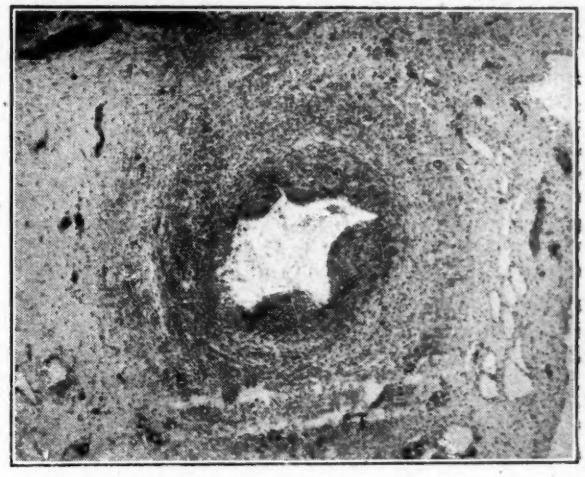


FIG. 1

varies with the state of the vessel. In structure and appearance they resemble the ridges found in the penile and uterine arteries (Kiss, 1921) and the pulmonary artery and ductus arteriosus (Pfeifer, 1902). They are found throughout the intra-abdominal portion of the umbilical artery from approximately the level of the base of the bladder to the umbilicus. It is of some interest to note that Jankovich (1939) described somewhat similar structures in the extra-abdominal portion of the artery. The function of the ridges appears to be to bring about a fairly complete closure of the umbilical arteries following birth—the circular muscle of the tunica media contracting and driving the ridges into the lumen.

With regard to the vein, the muscle fibres in the vessel wall are relatively deficient compared with those in the artery. The circular muscle fibres are found in scattered bundles and do

not form a complete ring. Although there are ridges projecting into the lumen they are poorly developed and deficient in muscle fibres (see Fig. 2). Connective tissue is much in evidence.

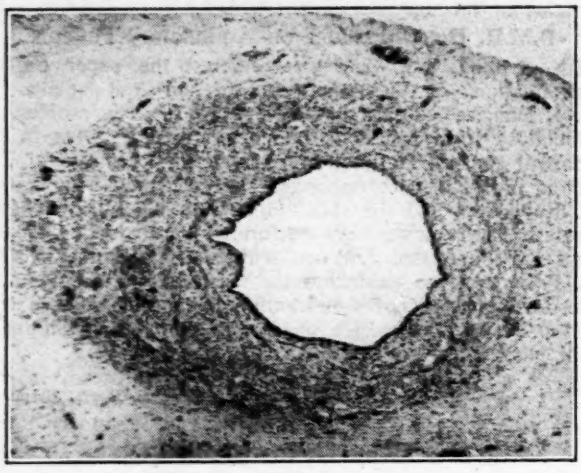


FIG. 2

The closing mechanism—namely, circular muscle and longitudinal ridges—is thus poorly developed in the umbilical vein. It should be noted, however, that Barclay, Franklin, and Prichard (1942), as a result of investigating the ductus venosus by means of radio-opaque substances, have demonstrated in certain animals a closing mechanism in the duct at its commencement.

There is no doubt, therefore, about the existence of a closing mechanism in the umbilical vessels, and although that in the vein does not appear very marked the mechanism in the ductus venosus may make up for this deficiency. In the African natives described by Dr. Price this closing mechanism in the umbilical vessels seems to function satisfactorily, but this is not always so. In this country, bleeding from an umbilical stump, even after it has been ligated, is not uncommon, and occurs at least often enough to warrant the suggestion in some textbooks that a second ligature should be applied to the umbilical-cord stump. Even in Rachmanow's cases, quoted by Dr. Franklin, although he found the cord did not need to be tied in 83% of his 10,000 patients, it had to be ligated in the remaining 17%—and that is not exactly a small number. One could perhaps account for Dr. Price's findings by racial difference. All one can say is that in the umbilical vessels a closing mechanism does exist, but this mechanism cannot always be depended upon. It would seem advisable, therefore, to continue ligating the umbilical-cord stump.—I am, etc.,

Department of Anatomy, University of Glasgow. IAN W. MONIE.

REFERENCES

Barclay, A. E., Franklin, K. J., and Prichard, M. M. L. (1942). *Brit. J. Radiol.*, **15**, 66. (Rachmanow, A. N. (1914). *Zbl. Gyräk.*, **38**, 590.)
Jankovich, L. (1939). *Disch. Z. ges. gericht. Med.*, **31**, 174.
Kiss, F. (1921). *Z. Anat.*, **61**, 455.
Pfeifer, B. (1902). *Virchows Arch.*, **167**, 210.

Perineal Tears in Normal Confinements

SIR,—I have been medical officer to an emergency maternity hospital since September, 1940, and I have been able to note the number of perineal tears which occur and which require one or more stitches. This hospital deals only with normal confinements, and the figures given below are for cases attended by midwives only; forceps cases are not included. I have personally dealt with all tears.

In the first 1,000 cases there were 236 tears: primiparae 191, multiparae 45; approximately 23.5%. I was surprised at this figure, as in private work the number of cases in which a doctor is called in seems very small. Through the kindness of Dr. A. E. Tibbitts, medical officer of health for Notts, I have obtained the percentage of medical aid notices sent in by midwives in 1,000 cases. The figure is 11.3%, and this includes all cases which require assistance—i.e., malpresentation, obstructed labour, etc.—and not only for perineal tears.

The only conclusion one can come to from these figures is that there are a considerable number of cases with perineal tears in domiciliary maternity work which are left without attention.—I am, etc.,

Retford. H. E. HUMPHRYS.

Infant Feeding

SIR,—In my work as a public vaccinator I have been impressed by the frequency with which I have come across mothers who complain of the fretfulness, tendency to prolonged crying, and failure to settle after feeds of their infants. My first inquiry in these cases almost always obtains the same answer. What quantity of milk is given at each feed? With breast-fed babies the reply is usually "Twenty minutes"; if the bottle is used it is "six ounces" or "seven," etc., as the case may be. In other words, the mothers have had it impressed upon them that baby should be given a fixed amount based on its age.

There are two reasons why this impression is made on mothers in clinics by nurses and health visitors, and even a paediatrician who wrote in your columns recently: (1) No allowance is made for the fact that a baby's appetite may vary, and vary it certainly does. (2) The bogy of overfeeding. Now it follows that if we are to make allowance for the fact that a baby's appetite varies we can only do so by giving the baby as much as it wants at each fed, and the invariably excellent results of so doing are sufficient proof that this practice is based on sound common sense.

Provided the quality of the milk is correct, the natural act of regurgitation in a greedy baby makes overfeeding impossible, and another cause of any symptoms such a baby may have should be sought. I believe that a very great advance in infant feeding, to say nothing of preventive medicine, would be achieved if all concerned would base their advice to mothers on these fundamental common-sense facts, and if the manufacturers of milk powders could be persuaded to remove from their tins, etc., the columns of figures linking the age of the child with fixed volumes of milk, leaving only details of the proportion of powder to water to ensure the correct mixture.—I am, etc.,

Winfrith, Dorset. P. R. BOUCHER.

Milk for School-children

SIR,—In your annotation on nutrition and the war (July 15, p. 84) you state, apparently on the authority of Dr. V. P. Sydenstricker, that "growing school-children, however, do not get enough first-class protein—youngsters of 6 get only half a pint of milk daily." The first contention may or may not be true, but a child of 6 can get half a pint of milk daily on his household ration and, in addition, one-third or two-thirds of a pint of milk at a reduced price under the Milk-in-schools Scheme, of which 76.4% of elementary-school children avail themselves.—I am, etc.,

Board of Education, S.W.1. J. ALISON GLOVER.

Estimation of Dental Caries

SIR,—Dr. Dorothy Smith implies (July 15, p. 94) that because Lady Mellanby has shown that caries detectable by probe and mirror alone has decreased since 1929 the investigation should not have been undertaken. Such criticism is as ungenerous as it is invalid.

The Dental Board, with an income of £20,000, devotes £2... to dental research. The official journal has not even commented on a paper of far-reaching significance to the dental profession.

Criticism from Oxford of the methods of a distinguished layman in the field of dietetics and dental research is singularly misplaced.—I am, etc.,

Blackheath. GUY NEELY.

Stomatology

SIR,—Much is being written about the practice of medicine and the training of the medical student, but nothing so far has been said about that increasingly important branch of medicine which, if I may use the term better known on the Continent than here, comes under the heading of stomatology.

In Continental usage this term designates the study of the abnormalities and diseases of the mouth, including the teeth, as a positive branch of medicine and not as a superspecialty of dentistry.

I hope that the respective findings of the Goodenough and the Teviot Committees will be so co-ordinated ultimately as to incorporate stomatology as a recognized study within the faculty of medicine.

Machinery for producing many more practitioners in this branch is especially necessary at the present juncture when there is a move to simplify the dental training and, in some quarters, a strong desire to separate dentistry from medicine.

From stomatologists should be drawn the appropriate consultants in a fully organized social health service, and it would be to them that the public might justifiably look for productive research in the biological complexities of the prevention of dental diseases.—I am, etc.,

London, W.1. C. BOWDLER HENRY.

Pentothal Anaesthesia

SIR,—The correspondence in your columns on the administration of pentothal, started by Dr. Montuschi (May 27, p. 731) and followed by letters from Dr. Sandiford and myself (June 17, p. 825), was originally intended to help many relatively inexperienced anaesthetists, in the Forces and on the Home Front, in the use of this drug. In your issue of July 15 (p. 93) you publish two letters—one from Mr. Thacker Neville and the other from Dr. Oswald Murphy. The only point that these two letters have in common is that they both apparently stray from this original intention. Mr. Thacker Neville's letter I consider to be most dangerous for the following reasons:

1. Operations on the nose and throat, in which there is a possibility of blood, pus, mucus, or other debris in the pharynx, are very likely to be complicated by laryngeal spasm, and the inexperienced anaesthetist should, except for induction, avoid pentothal, which is especially liable to this complication. Mr. Neville makes no mention of any packing, posture, or suction. Some or all of these are essential to prevent aspiration of debris or initiation of laryngeal spasm.

2. He considers picrotoxin to be "far superior to oxygen." Picrotoxin is a very potent convulsant, and it is only indicated in gross overdose of barbiturate. I have never had to use it during anaesthesia, although I always have it handy and have used it successfully in cases of attempted suicide by barbiturates. Maintenance of a clear airway, careful dosage, and the liberal use of oxygen, by controlled respiration where necessary, should avoid the need for picrotoxin.

3. Mr. Neville's fourth precept—never to give as much as morphine gr. 1/4—is not in accord with his self-confessed practice. A few days ago he did a septum, Caldwell-Luc, sphenoidectomy, and tonsillectomy all on the same patient, who had received morphine gr. 1/4 as part of his premedication.

4. I can conceive of no pathological reason why painting the face with industrial spirit should cause the death of a woman inadequately anaesthetized with pentothal. Such unsubstantiated bogies cause an air of witchcraft and voodoo to attend the administration of anaesthetics, which should be a rational procedure. Such bogies, once started, are very difficult to track down. Here is one at its source and it should be promptly exorcized.

5. Nowhere in his letter does Mr. Neville mention any scheme of dosage, so his extra notes do not add anything to Dr. Sandiford's most helpful advice.

Dr. Murphy, on the other hand, errs on the side of extreme caution. Much of his advice is a counsel of perfection, which we are unable to attain under war conditions.

1. There are not enough experienced anaesthetists available to restrict the use of this valuable drug to them alone. But they can, by helpful advice and sound example, save many a patient his life and many an occasional anaesthetist the unpleasantness of discovering the snags for himself.

2. Pentothal, *in suitably small doses,* is excellent for shocked, toxic, or debilitated cases; but the anaesthetist must know how to ascertain the correct dose, and in such cases the inexperienced should use the anaesthetic agent he knows best.

3. I agree that the strength of pentothal solution should never exceed 5%, and should be weaker for more serious cases.

4. An assistant, if available, is useful to hold up the jaw, but this act does not necessarily preclude the possibility of laryngeal spasm.

5. Isn't Dr. Murphy's rate of injection rather too slow? At this rate he must have been injecting for 20 minutes to induce anaesthesia in the second case he quotes. Pentothal is excreted so rapidly in the resistant patient that if injection is very slow the patient is excreting it as fast as it is injected.

Finally, I would suggest that pentothal anaesthesia is not only "not always child's play," it is never child's play. I tell my students that there is no such thing as "fool-proof" anaesthesia, but fools should not attempt to give anaesthetics.—I am, etc.,

London, W.1. F. W. ROBERTS.

Sodium Pentothal in Nasal Operations

SIR,—While reading Mr. Thacker Neville's account of his only death under pentothal anaesthesia (July 15, p. 93) I was arrested by the statement of his anaesthetist that death was due to the application of industrial spirit to the face (hardly a severe surgical stimulus) during the induction of anaesthesia. In the absence of any mention of the occurrence of laryngospasm, rightly or wrongly I gained the impression that death was considered to be due to primary cardiac failure. I should welcome the opinion of other anaesthetists as to the possibility of such an occurrence. In my opinion, when using pentothal in nasal surgery, the most important safety factor is efficient post-nasal packing to prevent any blood reaching the larynx and setting up laryngospasm. I should also like to stress the futility of anything except the gentlest artificial respiration in severe cases of laryngospasm until the cords relax, when the administration of oxygen under pressure is life-saving. Finally, in cases of cessation of respiration due to pentothal overdosage, relative or absolute, while not denying the beneficial effects of intravenous picrotoxin, I should, after the adoption of the Trendelenburg position, place rhythmical inflation of the lungs with oxygen under pressure as the most certain therapeutic measure, and would go so far as to say, "Never use pentothal unless there is at hand some means of carrying this out."—I am, etc.,

E. R. DINGLE,
Hon. Anaesthetist, Darlington Memorial Hospital.

The Cold Agglutinins

SIR,—I have read with interest the paper by Dr. Currie and your leading article on cold agglutinins (July 1, p. 16). These agglutinins may be either specific or non-specific. Auto-agglutinins usually belong to the latter class, and those which are active at greater than room temperature have been found in association with many morbid conditions, but rarely occur in the blood of healthy persons.

One of the most important features of autohaemagglutination is the "critical temperature" at which the antibody fails to react with the patient's cells. I have seen auto-agglutinins associated with haemolytic anaemia on three occasions, and in all these cases the critical temperature was greater than 30° C. The many other instances of autohaemagglutination which I have studied have not shown a critical temperature as great as 30° C., nor has any case been one of haemolytic anaemia. I therefore attach considerable importance to this feature, which seems to be of more significance than, for example, the antibody titre.—I am, etc.,

Manchester. F. STRATTON, M.D.,
Regional Transfusion Service.

Intravenous Barbiturates in Hysteria

SIR,—Psychiatrists will read with some interest the article by Drs. Lambert and Linford Rees (July 15, p. 70) on the treatment of 247 cases of hysteria by three separate methods—intravenous barbiturate, general psychiatric treatment, and hypnosis. The interest, I think, will not reside so much in the fact which emerged that there were no significant differences in the results of the three forms of treatment. The more important, not to say startling, fact is that mere symptomatic recovery is accepted by the authors as the adequate test for evaluating the results. "The criterion of return to duty as an indication of therapeutic success was found to be unsatis-

factory" mainly, apparently, because it implied the need for a total assessment of the case and not merely improvement in the symptoms.

With an easy therapeutic target of this kind, spectacular successes are to be expected. In fact, from my experience, I would say it is pretty certain that equally good results would be obtained in a similar group of cases if they were given no special treatment, but simply told that they would not be sent back to duty if they recovered from their symptoms within a specified time.

We evidently had higher therapeutic aims in the last war, and I suggest that the article would have been somewhat less misleading if the authors had referred not to the treatment of hysteria, as they do, but to the treatment of the symptoms of hysteria.—I am, etc.,

London, W.1. FREDERICK DILLON.

Chemotherapy in Otitis Media

SIR,—I read with great interest the article by Mr. Dingley on the dangers of sulphonamide therapy in otitis media (June 3, p. 747) and with astonishment some of the correspondence which has followed. I am quite sure that neither by word nor by suggestion did Mr. Dingley propose the abolition of sulphonamides in all types of middle-ear infection, but what he was emphasizing was that chemotherapy was not, in itself, a substitute for myringotomy in a case where pus was present behind the drum. With this contention I find myself in hearty agreement. That sulphonamide therapy in early cases of otitis media will hasten their cure and thereby reduce the number that would doubtless have gone on to a mastoid infection will, I think, be a matter for general acceptance. The response is often dramatic; but I do feel that when pus is present behind a drum, that drum is safer and better opened, and there is more chance thereby of avoiding the mastoid operation than by pouring in sulphonamides, even in full doses.

I agree, too, with Mr. Dingley in his warning that the sulphonamides in such cases can mask symptoms. I have seen several, and the lesson to be learnt is, I think, that while sulphonamides do achieve great things it is not reasonable to expect them to do everything, least of all finally to sterilize and render innocuous pus that is behind the ear-drum.—I am, etc.,

Cheam, Surrey. C. CARRON BROWN.

Mastoiditis and Mastoidectomy

SIR,—I have carefully and with deep interest read Mr. W. H. B. Magauran's letter (July 1, p. 23) in answer to mine of March 18 and May 6. He states that he has performed during recent months 97 conservative mastoid operations. Conservative! I cannot conceive of any less conservative method of treating an ear case than by mastoidectomy. If this is Mr. Magauran's idea of the meaning of conservative, then he and I are poles apart, and I cannot accept his definition of the words dangerous, disfigurement, pain, and good or bad results.

Mr. Magauran will agree that the majority of the cases he operates upon are those typical ones described in every textbook as cases of otitis media which require the operation of mastoidectomy. This, I again state, I have through years of observation and as the result of my experience since 1912, and more especially since 1919, come to disagree with entirely, and to form the opinion that the teaching for the last forty years or so on this subject has been wrong and disastrous to the public.

The 14 cases which Mr. Magauran cites are remarkably like those I quoted as following mastoidectomy: (1) *General streptococcal meningitis.*—I find that has been the most frequent cause of death after mastoidectomy. (2) *Meningeal reactions with turbid C.S.F.*—Cause similar but much less reaction. (3) *Otitic hydrocephalus and subdural abscess.*—Caused by chiselling in a septic cavity and removing the bony barrier separating it from the brain. The other cases—lateral sinus infection (he missed out infection of the jugular vein), external rectus paresis, facial paresis, vertigo, extradural abscess, etc.—have all been seen by practitioners to have followed mastoidectomy. Bezold's abscesses have been opened frequently and the bone at the tip of the mastoid found cupped from the outside but hard and sound when the aural surgeon has gone on to open the mastoid bone, proving that the infection comes from outside and has not penetrated through this very hard bone. As to painful dressings, the patient is the one to answer that; the distress is not easily forgotten.

I am glad Mr. Magauran agrees that those cases which he calls "mastoiditis" and I call "otitis media" can be cured conservatively. He states: "In a series of 132 cases of acute mastoiditis treated in one hospital complete resolution occurred in 77 cases with conservative treatment. . . . The remaining 55 cases had mastoid operation either immediately or after conservative treatment had failed." Mr. Magauran thus differs from his fellow aural surgeon, Mr. Gordon Mowat, who, in the *Journal* of April 8 (p. 512), in answer to my letter of March 18, writes: "Almost any case of mastoiditis will subside without operative treatment, but, nevertheless, infection of the mastoid is present, and the ears continue to discharge." Mr. Mowat states his belief that all cases of discharging ears should have mastoidectomy performed within six weeks of the discharge commencing. He does not say how soon he would operate, whether immediately or on the last day of the six weeks. Nevertheless he states that mastoiditis is never cured except by mastoidectomy. I, on the other hand, find that all those cases can be cured by a very simple conservative treatment. I maintain that if those of the 55 cases cited by Mr. Magauran as having had the mastoid operation performed immediately had been given a chance of conservative treatment, and those which were operated upon after a period of conservative treatment had been treated as I have advised, the whole 132 cases would have completely recovered—not just 77.

Mr. Magauran and his fellow aural surgeons would earn the undying gratitude of a multitude of sufferers if they would agree to try proper and careful conservative treatment, say, for the next six months. The tools required are few—a small bottle of hydrogen peroxide, a small bottle of methylated spirit, some cotton-wool, and a pair of fine aural forceps. The other requisites—one of the emollient types of drops ("sedonan"), aspirin and phenacetin powders, an inhalant—are not so immediately necessary. The time taken is not long. I spend about half an hour on my first visit and ten minutes or a quarter of an hour on my subsequent ones. I visit for about three days running and then once a week. My average time for a cure is six weeks—i.e., about nine visits. Some cases clear up remarkably quickly, and often those with the most alarming symptoms of pain, pushing forward of the ear, etc. Others have a fairly harmless watery discharge for many weeks, but know how to treat themselves, and only call to see me once in a while to be reassured that all is going well. The pain I practically stop on my first visit. In fact I do not leave the house until the patient smiles and says there is little or no pain, and in small children generally when they have fallen asleep after relief from pain.

Mr. Magauran's misgiving is very natural; it is a good sign. It was my misgiving after carefully watching aural surgeons operate and seeing young and healthy friends and relatives die or have a great struggle to recover from mastoidectomy that made me refuse ever to have a patient of this large practice operated upon for "mastoid," and the results, as I state, have been uniformly excellent. Treatment with such results for 25 years is surely worth a real trial. Courage, patience, and care are all that is required. The first is very necessary after we have all been taught just the reverse of what I have stated. I advise doctors, if they do have to have a patient operated upon for otitis media, to go to the theatre and see the crude, unscientific operation it is and ask themselves seriously, Is this operation necessary and is it a safe one?—I am, etc.,

Edinburgh. H. HILTON BROWN.

Sulphonamide Prophylaxis of Gonorrhoea

SIR,—It was most surprising to read (May 20, p. 695) that responsible authorities had used sulphonamides in the prophylaxis of gonorrhoea. Here in the Gold Coast there has never been any effective control of the sale of these drugs to the public. As everybody gets gonorrhoea they soon became very popular, and for years now the people have been

dosing themselves with tablets indiscriminately, not only for gonorrhoea but for every kind of complaint, even using them as a "pick-me-up."

In times of scarcity the price has risen to a shilling a tablet; forged tablets have appeared on the market; and professional thieves have found stocks of sulphonamides more attractive than tills. Thus conditions have been ideal for the development and spread of resistant strains. Already in the larger centres a high proportion of gonococcal and puerperal infections fail to react to a sulphonamide; it may very soon become quite useless.

Thus to throw a valuable drug to the wolves of commerce seems an inexcusable insult to medical science. Will penicillin share the same fate?—I am, etc.,

Koforidua, Gold Coast. G. L. ALEXANDER.

Gonorrhoea in North Africa

SIR,—The cases described by Col. Campbell are typical of those we used so frequently to come across in the days of irrigation treatment. As I pointed out some ten years ago the sequelae of post-urethral and epididymal troubles are probably due to irrigation with lodgment of the infection in the seminal vesicles. When this occurs vasotomy—as I then advocated—is the only treatment which will bring about a cure. This also holds good to-day for all cases in which sulphonamide treatment fails. If Col. Campbell will treat his cases by vasotomy he will find that they will not require to remain in hospital for more than three to four days, by which time all discharge and symptoms will have disappeared and the patient be fit for duty.—I am, etc.,

Sandbach, Cheshire. J. F. PEART.

Stilboestrol in Cancer

SIR,—On my return from a holiday I was interested to read your annotation (July 1, p. 17) wherein you stated that Huggins of Chicago was the first to suggest that stilboestrol might play an important part in the treatment of malignant disease, and that you considered the evidence is strong that its mode of action is by inhibiting the gonadotrophic hormone of the anterior pituitary.

I am unfortunately not in a position here to find out when Huggins thought of it, but may I with all due humility point out that I, quite unaware of Huggins's work, also suggested this treatment in a letter to the *Journal* of July 27, 1940 (p. 133), mentioning that stilboestrol most probably had an inhibitory effect on the secretion of the anterior pituitary, and quoting R. L. Noble's experiments in support of this theory. Do please credit us with some imagination in Britain as well as the Americans.

The use of stilboestrol in cancer is full of promise, and I am pleased to know that the clinical experiments are to be continued.—I am, etc.,

Upholland, Wigan. J. THOMSON SHIRLAW.

Tarso-metatarsal Dislocation

SIR,—It was with great interest that I read Surg. Lieut. Dorrell's account of a case of tarso-metatarsal dislocation in your issue of July 1.

I have in course of preparation an article describing five cases of this dislocation which it has fallen to my lot to treat during the last 2 or 3 years. In three of these the injury has been almost identical with that described by Mr. Dorrell—in two the foot was crushed by the landing-wheel of an aircraft and in the third by the wheel of a double-decker bus. In each case the vehicle was moving very slowly (the aircraft were being towed into a hangar and the bus was reversing), and I agree that it is the slow crushing of the foot which produces the dislocation. My treatment was similar to Mr. Dorrell's, but the bus injury required skeletal screw traction with wires through the calcaneus and metatarsal heads. All the other cases reduced easily by manipulation with a distinct click.

As Mr. Dorrell points out, the immediate prognosis is good but late prognosis is problematical. I have been unable to follow up my cases for longer than 6 months.—I am, etc.,

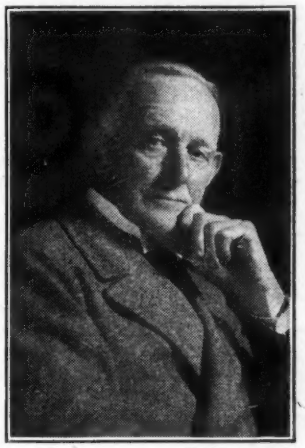
Ulverston. DAVID A. RICHMOND, Surgeon, E.M.S.

Obituary

SIR HUGH RIGBY, Bt., K.C.V.O., F.R.C.S.

We regret to announce the death on July 17 of Sir Hugh Rigby, Serjeant-Surgeon to the King from 1928 to 1932, and consulting surgeon to the London Hospital and the Poplar Hospital for Accidents. In 1922 he operated on the late Duke of Kent, then Prince George, for appendicitis; and it was he who on Dec. 12, 1928, performed at Buckingham Palace the operation for empyema which was the turning-point in King George V's very severe and protracted illness.

Hugh Mallinson Rigby was born in Dublin on May 19, 1870, son of John Rigby, M.A., late superintendent of the Royal Small Arms Factory at Enfield. He was educated at Dulwich College and at University College, London, and studied medicine at the London Hospital, where he soon made his mark as a man of great promise. He graduated M.B.Lond. in 1895, B.S. (with first-class honours and gold medal) in 1897, and M.S. in 1901; and obtained the F.R.C.S.Eng. diploma in 1900. At the London Hospital he held a succession of junior posts: house-surgeon and house-physician immediately after qualifying, and then surgical registrar, demonstrator of anatomy and tutor in elementary clinical surgery, and demonstrator of clinical pathology. He was elected assistant surgeon to the hospital in 1902 and later became full surgeon. During the last war he served, with the rank of colonel, A.M.S., as consulting surgeon to the B.E.F. in France and to the London District. His services were mentioned in dispatches and he was created K.C.V.O. in 1918.



[Elliott and Fry

Sir Hugh Rigby was appointed surgeon-in-ordinary to the Prince of Wales and surgeon to the Prince of Wales's Household in 1923, and Serjeant-Surgeon to the King in 1928. He was created a baronet in June, 1929, and became Honorary Surgeon to the King in 1932. He retired from the active staff of the London Hospital before reaching the age limit and was elected consulting surgeon, but he continued in private practice for some years. In addition to his duties at the London and the Poplar Hospitals he has been surgical consultant to the City of London Maternity Hospital, to the East Ham Hospital, and to a number of cottage hospitals in the neighbourhood of London. In 1931 he received the Honorary Fellowship of the Royal College of Surgeons in Ireland, and two years later the honorary degree of M.Ch. from the National University of Ireland.

When he finally gave up active work Sir Hugh Rigby went to live at his country home, Long Durford, Petersfield, Hants. Throughout his career he was essentially a working surgeon, sound in judgment and in action, inspiring the confidence of all who served him and all whom he served. He avoided publicity and left medical politics to others. His writings were few: an occasional paper on a surgical subject and the article on diseases of the oesophagus in *Choyce's System of Surgery*.

SIR WILLIAM LISTER

We have received the following appreciation from H. B. S.:

As one of the younger generation of eye surgeons I wish to pay a tribute to Sir William Lister, whose example as an eye surgeon was quite admirable. Those of us who attended his clinic and his operations at Moorfields will always remember him for his humanity and kindness in handling patients, his intense enthusiasm for ophthalmology, and his exquisite skill as an operator. In the theatre his behaviour gave us a perfect example of the qualities that make a great surgeon: imperturbability, a high degree of technical skill

which never varied whatever the circumstances, and a quiet orderly manner which gave confidence to the patient and the theatre team. His grace and his unhurried ways were good to see. His few courteous words to a patient on entering the theatre to undergo the ordeal of an eye operation under local anaesthesia never failed to give reassurance, and when the operation was over and the dressings applied a gentle tap on the shoulder and a quiet voice saying, "All has gone very well and you have behaved admirably," did much to induce that state of contented composure so necessary after an eye operation.

As a teacher he was gifted with dramatic talent in describing a case. So vivid were these accounts of his clinical experiences that their memory is quite indelible. Some of us, now 15 years or more after he taught us, can recall fully the contusion in the eye of the Charterhouse boy keeping wicket and the complications which befell the fine cavalry officer suffering from basal-celled carcinoma of an eyelid, and others.

He belonged to a generation of great men in the field of ophthalmology. For those of us who have had the privilege of being taught by him his memory will remain ever fresh in our minds as a most courteous and gallant gentleman, a surgeon of the highest quality, and a teacher whose enthusiasm has given us a lasting inspiration.

Dr. ROGER NELSON WILCOX, medical officer of the East African Medical Service, died on April 10 at Salisbury Hospital, Southern Rhodesia, after an illness of two months. Born in 1903, the younger son of George Wilcox of Ealing, London, he was educated at St. Paul's School and received his medical education at St. Thomas's Hospital, qualifying M.B., B.S.Lond. in 1929, and obtaining the D.L.O. in 1930. After holding the posts of house-surgeon to the ear, nose, and throat department at St. Thomas's he then became house-surgeon at the Kent and Canterbury Hospital. Joining the Colonial Medical Service after a course at the Tropical School, where he obtained his D.T.M.&H., he was posted to Nyasaland in 1931, where he served for 13 years and continued there until his death. He was a very keen member of the B.M.A., and attended all meetings of the Nyasaland Branch, whose president writes: In harness until his last illness, Wilcox's premature death is deeply mourned by his many hundreds of friends in Nyasaland. He was beloved by all his patients, who were quick to realize that his shy manner on first acquaintance was merely superficial. As well as his great service to the Europeans in Nyasaland as medical officer in charge of Blantyre European Hospital he also worked for many years for the African community, and he was stationed at Port Herald, Karonga, and Kota-Kota Districts. He is deeply missed by his African staff and by all his Indian and African patients. He was a very able surgeon and specialized on the ear, nose, and throat. In particular, his old friends in Blantyre and his colleagues in Nyasaland and the district officers with whom he was stationed feel his passing has left a great gap. He was an excellent diagnostician and very kind-hearted with all with whom he came in contact. He was a wonderful listener and had a very keen sense of humour, and was a staunch and loyal friend. He was also a keen sportsman, playing an excellent game of tennis and golf.

News has reached England of the death of ROSS MILLAR, M.A., M.D., C.M., at Ottawa on June 15. Dr. Millar will be remembered by many of the medical officers of the R.A.M.C. in the Third Army during the 1914–18 war, where he served for two years with No. 37 C.C.S., and for the final year of the war with No. 9 C.C.S. as surgical specialist. After the close of the war Dr. Millar returned to his home in Nova Scotia, where he resumed his private surgical practice and was elected a Fellow of the Canadian College of Surgeons. In 1928 he was appointed Director of Medical Services in the Dominion Department of Pensions; and he moved to Ottawa, where he resided until his death. He joined the Halifax Branch of the B.M.A. in 1906 and remained an enthusiastic member of the Association after its Canadian local units were disbanded.

Dr. FRANCIS WHITE HOPE ROBSON, who died on July 2 at Bournemouth, received his medical education at Edinburgh University and the Royal College of Surgeons, Edinburgh. He graduated M.B., C.M. in 1893, and in 1900 took the M.D. with honours; he also held the Medico-Psychological Certificate. After a few years in house-surgeon appointments and four years in a country practice he settled in Southampton and had a busy practice there for 32 years. He held an appointment as public vaccinator for the Shirley district, and also acted as test lymph vaccinator in Southampton for the Ministry of Health. He was for 15 years medical officer to the Southampton Corporation children's homes. He took an active part in Southampton medical work, and was secretary to the Panel Committee for five years after the National Health Insurance Act came into force; he was also on the committee and acted as treasurer to the local public medical service when Southampton played a leading part and was among the first to enter the fight in what was known at that time as the Battle of the Clubs. He also was a member of the Southampton Insurance Committee, Tuberculosis Committee, and the Medical Service Subcommittee. Hope Robson retired to Bournemouth in 1936. He was a member of the B.M.A. for over 40 years. He invented a simple aspirator which could be operated with any ordinary syringe, and so eliminated the need for using a suction pump.

Dr. FREDERICK HENRY died at his home in Oldpark Road, Belfast, on July 5, aged 43. He was the second son of the late Major Robert Henry of the Indian Army, and qualified M.B., B.Ch., B.A.O. of the Queen's University of Belfast in 1927. After some experience in general practice in Liverpool and in Glamorganshire he returned to Belfast and gained for himself a growing practice and the confidence and affection of his patients and his colleagues. He dearly loved his work and left nothing undone to elucidate a problem or to relieve suffering.

"C. G. L." writes: A host of doctors will learn with sorrow of the death of Mr. A. HARDES, who for 30 years was with what is now the British Medical Bureau, where he managed the "locumtenens department." Here he was the intermediary between thousands of "tenentes" and the territorial owners of "loci," and to his task he brought gifts of kindliness, helpfulness, insight, and tact. To many he was but a voice over the telephone, but his personality came over the wire in the same way as the impact of an actor's personality may come over the wireless.

The Services

Capts. E. J. Bowmer, D. H. McCollum, and F. Zangen, R.A.M.C., and Capt. T. Stephens and Lieut. T. G. Jones, I.A.M.C., have been awarded the M.C. in recognition of gallant and distinguished service in Italy.

CASUALTIES IN THE MEDICAL SERVICES

Killed.—War Subs. Capt. J. D. Laurie, R.A.M.C.
Died.—Major R. J. Franklin, R.A.M.C.

DEATHS IN THE SERVICES

By the death on July 15 at Birnam of Col. FREDERICK PERCIVAL MACKIE, C.S.I., O.B.E., M.Sc., M.D., F.R.C.P., F.R.C.S., I.M.S., that service has lost one of its most distinguished members at the age of 69. He was educated at Dean Close School, Cheltenham, at the Bristol Medical School and St. Bartholomew's Hospital, and qualified in 1897. In 1902 he passed the F.R.C.S.Eng. and in the same year gained first place in the entrance examination for the I.M.S. and won prizes at Netley. In 1903 he was medical officer to the Younghusband mission to Tibet and in 1905 entered the Bacteriological Research Department as assistant director of the Plague Research Laboratory, Bombay. Among other important research appointments he was a member of the Uganda Sleeping Sickness Commission in 1908, and special research officer on kala-azar in 1911. Much of the research work he carried out in Bombay was on plague, but his most noteworthy discovery was that the spirillum of relapsing fever, which is common in Bombay, is transmitted by the bites of body lice. He was also associated with Col. Hamilton Fairley in important work on the dietetic treatment of sprue and also worked at dysenteries, enteric fever, and schistosomiasis. During a period of leave he passed the M.R.C.P. and in 1919 was elected to the Fellowship for his researches; he became an M.Sc. Bristol in 1916 and D.P.H. in 1925. He also saw military service in Baluchistan and served during the war of 1914–18 when he was twice mentioned in dispatches and received the O.B.E. In 1920 he held the professorship of pathology at Calcutta for a short time; in 1921 he became Director of the Pasteur Institute in Assam; and in 1923 he was appointed Director of the Haffkine Institute, Bombay, where so much of his earlier work had been done. Among senior administrative posts he officiated as Public Health Commissioner, Government of India, in 1928, Surgeon-General with the Government of Bombay in 1929, and Director of the Pasteur Institute and Research Laboratory, Shillong, in 1930. He also held the appointments as Honorary Surgeon to H.M. the King and to the Viceroy of India. On his retirement under the age rules Col. Mackie soon found scope for his abilities, first as pathologist at the London School of Tropical Medicine and soon after as chief medical officer to the British Overseas Airways Corporation, and he worked at the fumigation of planes to destroy

yellow-fever-carrying mosquitoes. His great abilities and painstaking and most reliable work placed him in the forefront of medical research workers in India.

Correction

A typing error in the obituary notice of Capt. S. C. H. Hood (July 15, p. 97) gave the date of his death as July 13 instead of June 13.

Universities and Colleges

UNIVERSITY OF MANCHESTER

At the last meeting of the Council the Vice-Chancellor announced the offer of £5,000 from the Governors of the Royal Manchester Children's Hospital towards the foundation of a Child Health Institute. This offer is the first promise received since a conference was recently held in the University to consider the establishment of an Institute of Child Health. The conference was attended by representatives of some of the local authorities, the voluntary hospitals, and other institutions interested in child health. The Vice-Chancellor explained that the cost of a professorial department in this subject would be from £7,000 to £10,000 per annum, and that the establishment of the department would depend on the provision of the necessary funds from outside sources. The proposal had been met by a most encouraging response from those present at the conference, who had promised to raise with the various bodies they represented the question of annual grants for this purpose.

Dr. G. Bridge of Preston has made a gift of £10 per annum, to provide a prize in obstetrics in the Final M.B., Ch.B. examination.

UNIVERSITY OF SHEFFIELD

The Council of the University received on July 14 from Sir Arthur Hall, M.D., F.R.C.P., a volume of personal reminiscences and an album of photographs illustrating the development of the Sheffield School of Medicine since its inception. The Council accorded its warm thanks to Sir Arthur Hall for this gift.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

At a quarterly meeting of the Council held on July 13 Sir Alfred Webb-Johnson was re-elected President and Sir Girling Ball and Mr. C. Max Page Vice-Presidents. Mr. R. Milnes Walker was elected a member of the Court of Examiners for a period of three years from August 3 next.

The following appointments were made for the ensuing year:

Hunterian Professors.—Mr. Cecil A. Joll, one lecture on Conservative Surgery of Carcinoma of the Rectum and Recto-sigmoid; Mr. T. B. Layton, two lectures on the Inflammations of the Middle-ear Cleft: considered Clinically, Pathologically, and Therapeutically from the Individual and National Standpoints; Surg. Capt. Lambert C. Rogers, one lecture on Ligation of Arteries; Mr. D. M. Stern, one lecture on the Aetiology and Treatment of Prolapse, with Special Reference to Cystocele and Stress Incontinence; Major H. S. Shucksmith, one lecture on Abdominal Injuries in Battle Casualties: Clinical Aspects in 100 Personal Cases; Squad. Ldr. T. Cradock Henry, one lecture on Aviation Injuries of the Face; Miss Jean M. Dollar, one lecture on the Use of Plastics in Ophthalmic Surgery; Squad. Ldr. D. N. Matthews, one lecture on Storage of Skin for Autogenous Grafts; Mr. Andrew K. Monro, one lecture on the Treatment of Acute Appendicitis; Mr. James Patrick, one lecture on a Study of Supination and Pronation, with Special Reference to the Treatment of Forearm Fractures; Mr. A. S. Aldis, one lecture on Injuries of the Pancreas and their Surgical Treatment.

Arris and Gale Lectures.—Prof. Francis Davies, one lecture on the Early Development of the Human Embryo; Dr. D. V. Davies, one lecture on the Synovial Membrane and the Synovial Fluid of Joints; Mr. J. T. Chesterman, one lecture on Some Alterations of the Neuromuscular Balance of the Intestine and their Clinical Significance.

Erasmus Wilson Demonstrators.—Mr. L. E. C. Norbury, O.B.E., one demonstration; Mr. R. Davies-Colley, C.M.G., three demonstrations; Mr. C. E. Shattock, one demonstration; Mr. Timothy M Tyrrell, one demonstration with ophthalmic pathological material.

Arnott Demonstrator.—Prof. A. J. E. Cave, six demonstrations.

The fifteenth Macloghlin Scholarship was awarded to S. T. H. H. Pilbeam of Lewes County School. Dr. F. K. Sanders and Mrs. H. P. Herbert were re-elected Leverhulme Research Scholars. Votes of thanks were given to Mrs. Crile for a representative collection of works of the late G. W. Crile, to Dr. W. Loudon Strain for a gift of more than 100 books, and to Miss C. F. Thompson for pamphlets and medals from the collection of her father, the late C. J. S. Thompson.

Diplomas

A Diploma of Membership was granted to Peter Hansell.

Diplomas were granted, jointly with the Royal College of Physicians of London, as follows:

DIPLOMA IN PUBLIC HEALTH.—Leonora A. Crawford, C. E. Jamison, Susan M. Tracey, R. K. W. Yang.

DIPLOMA IN PSYCHOLOGICAL MEDICINE.—R. S. Allison, T. H. B. Gladstone, A. Goldberger, R. R. H. Mitchell, H. J. O'Loughlin, F. Reitman, A. M. Stewart-Wallace.

DIPLOMA IN LARYNGOLOGY AND OTOLOGY.—M. M. A. Cader, E. H. M. Foxen, C. E. Gardiner, A. G. Gibb, J. B. Sugden.

ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH

A quarterly meeting of the College was held on July 18, the President, Dr. A. Fergus Hewat, in the chair. Dr. J. Colin Caird (Edinburgh), Dr. T. A. MacGibbon (Thetford, Norfolk), Dr. T. McL. Galloway (Carlisle), Dr. W. D. Henderson (Woking, Surrey), and Dr. H. P. Tait (Edinburgh) were introduced and took their seat as Fellows of the College. Dr. W. F. Brown (Ontario, Canada) and Dr. R. Maxwell Johnstone, *M.C.* (Edinburgh) were elected Fellows.

CONJOINT BOARD IN SCOTLAND

The following candidates, having passed the final examination, have been admitted L.R.C.P.Ed., L.R.C.S.Ed., and L.R.F.P.&S.Glasg.: D. J. Alexander, G. A. F. Bain, J. Bates, W. A. Benson, J. M. Blyth, T. Bowden, A. A. Bradley, J. McD. Cairns, F. J. Clarke, T. Dibdin, F. F. Dougall, A. J. K. Finlayson, S. Freedlander, Joan E. Gray, S. Harris, J. Hendrie, Helen W. Kerr, A. S. Little, G. B. Macaulay, W. McIntyre, T. G. McKendrick, A. S. A. Monem, J. McC. Murdoch, A. J. Neil, W. A. Robson, S. R. Shaw, Dorian S. Smith, J. A. Smith, Fanny Stang, D. Sweeney, V. P. Vohra, J. T. Weir, J. H. White, H. W. Woolner.

Medical Notes in Parliament

White Paper Legislation

Sir HENRY MORRIS-JONES on July 20 asked the Minister of Health whether, in view of the general misgiving about the White Paper proposals on a National Health Service, expressed in recent months by many local authorities, voluntary hospital associations, and the medical and allied professions, he would postpone the introduction of legislation pending further discussion in the House, or until after a general election. Mr. WILLINK did not think that there was any general desire or that it was the wish of the House that the Government should depart from the procedure previously announced, which assumed a stage of full discussion with the professional and other organizations concerned, followed by the submission of legislative proposals.

Flying-bomb Casualties

On July 18 Sir HENRY MORRIS-JONES asked the Home Secretary when he intended publishing the number of casualties from flying bombs and what period the statement would cover. Miss WILKINSON referred to the statement covering the month of June which was published on July 14. Similar statements would be published for succeeding months. Replying to Mr. Douglas, Miss Wilkinson said that the figures for June related entirely to casualties caused by flying bombs; there were no casualties through piloted aircraft. On security grounds the Government thought it would be undesirable if they published separate figures for casualties from piloted and from non-piloted aircraft. It would tell the enemy too much. The figures covered all casualties from non-piloted aircraft, both Service and civilian.

Evacuation from London

From July 2 to July 20 over 170,000 women and children were evacuated from London in organized parties. Mr. Willink remarks that considering the large numbers involved and the speed of the operation, it has been effected with a smoothness that reflects credit on all concerned. Some difficulties have inevitably occurred, but the reception in the safer areas has on the whole been very satisfactory. He urges upon those who have been evacuated to safer areas that they should remain there.

Penicillin for Prisoners of War.—Sir JAMES GRIGG, replying on July 18 to Mr. Robertson, said that small quantities of penicillin were being sent through the agency of the British Red Cross Society to selected medical officers for the treatment of specified patients in hospitals in enemy countries where our prisoners were.

Claims for an Arthritis Treatment.—Mr. E. P. SMITH asked on July 13 whether the Minister of Health knew that arthritis had been successfully treated with "ertron," a derivative of ergosterol, at the Cook County Hospital, Chicago. Mr. WILLINK said his information was that the claims made for the product called "ertron" in the treatment of arthritis were not accepted by the most authoritative medical opinion in the United States, and that clinical evidence over some seven years past did not warrant the belief that it had beneficial effects.

Diagnosis of Smallpox.—Mr. WILLINK stated on July 14 that he had arranged to issue to all medical officers of health a statement showing what condition justified a diagnosis of smallpox, and what differences should be noted between a case of smallpox and other diseases.

Medical News

The Council of Epsom College will shortly proceed to award St. Anne's Scholarships to girls attending Church of England schools. Candidates must be fully 9 and under 16 years of age, and must be orphan daughters of medical men who have been in independent practice in England or Wales for not less than five years. The value of each scholarship is dependent upon the means of the applicant and the locality and fees of the school selected. Forms of application can be obtained from the Secretary's Office, Epsom College, Epsom, Surrey, and must be completed and returned by Oct. 15.

The American Association for the Advancement of Science will hold its annual meeting for 1944 at Cleveland, Ohio, from Sept. 11 to 16. Two previous annual meetings have been cancelled at the request of the U.S. Office of Defense Transportation.

A plan of the organization of medical services in UNRRA in both the European region and in Washington has just been issued. In the European region there is a standing technical subcommittee on health (chairman, Dr. Melville Mackenzie) comprising fourteen accredited representatives of the national health administrations of member Governments. This has three expert commissions: one on quarantine and one on health problems of displaced persons (chairman of both is Dr. P. G. Stock); and a third on nutrition under the chairmanship of Sir J. C. Drummond. The Health Division Staff of UNRRA includes: Dr. Andrew Topping (Director of Health), Dr. Neville Goodman and Dr. Dudley Reekie (Deputy Directors), and Drs. G. Stuart, G. Johnstone, F. Daubanton, and R. Coigny (medical officers). In addition the following have been appointed to the field staff (medical): Dr. J. J. Paterson, Dr. H. Stanley Banks, Dr. J. Balfour Kirk, and Dr. K. W. C. Sinclair-Loutit.

Dr. Walter B. Cannon, professor emeritus of physiology, Harvard Medical School, has been appointed visiting professor of physiology at the New York University College of Medicine.

The Army Council has given permission for Col. M. Stoddart-Scott, T.D., M.D., R.A.M.C., Creskeld Hall, Arthington, Yorks, to be announced as prospective Conservative candidate for the Pudsey and Otley Parliamentary Division of the West Riding of Yorkshire.

EPIDEMIOLOGICAL NOTES

Discussion of Table

In *England and Wales* during the week there were only small variations in the trends of infectious diseases. There were 57 fewer cases of whooping-cough than last week, and 47 of scarlet fever, but measles went up by 68.

Diphtheria notifications were 7 fewer than last week's low record. The largest decline in the incidence of scarlet fever was in Essex, where 30 fewer cases were recorded. The local trends of whooping-cough fluctuated: Essex had 84 fewer cases than last week, and Warwickshire 38; but Kent had 42 more, Gloucestershire 41 more, and Hertfordshire 40 more. There were 37 fewer cases of measles in Kent and 39 in Staffordshire, but 42 more in Wiltshire and 34 more in Essex. One-fifth of the total cases of measles were recorded in Lancashire.

Dysentery notifications numbered 132—3 fewer than in the preceding week. The increase in Glamorganshire from 12 to 25 cases was the only local rise of any size. The largest of the other returns were Lancashire 20, and Surrey 12.

In *Scotland* there was a general decline in the incidence of infectious diseases—96 fewer cases of measles, 48 fewer of whooping-cough, 47 of dysentery, 45 of diphtheria, and 23 of scarlet fever. Both diphtheria and dysentery were at the lowest level during recent months. Almost one-third of the total of diphtheria notifications, 29 out of 91, were recorded in Glasgow. The largest returns for dysentery were Glasgow 14, and Edinburgh 13.

In *Eire* the incidence of measles remained high. The largest return was from Clare, Ennis U.D., with 129 of the 210 cases. There was a small rise in the notifications of diphtheria and whooping-cough, three-quarters of the cases of the latter being in Dublin C.B. The 62 cases of diphtheria were spread over thirty-five registration areas.

In *Northern Ireland* the notifications of measles fell from 55 to 29, but those of scarlet fever rose slightly, from 45 to 52. Only 13 cases of diphtheria were notified, the lowest total for two months.

Week Ending July 15

The returns of infectious diseases in England and Wales during the week included: scarlet fever 1,394, whooping-cough 2,323, diphtheria 491, measles 2,536, acute pneumonia 437, cerebrospinal fever 59, dysentery 156, paratyphoid 7, typhoid 8.

No. 27

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended July 8

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1944 (a)	(b)	(c)	(d)	(e)	1943 (Corresponding Week) (a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	45	2	24	1	—	49	4	26	3	1
Deaths		1	1				1	—		
Diphtheria	425	12	91	62	13	580	45	118	59	30
Deaths	5	—	—	2	2	13	1	2	1	—
Dysentery	132	5	48	2	—	93	12	64	1	—
Deaths				—	—				—	—
Encephalitis lethargica, acute	1	—	1	—	—	2	—	—	1	—
Deaths		—					—			
Erysipelas			41	6	—			34	4	2
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				5					44	
Deaths	35	8	13	12	5	42	10	8	14	9
Measles*	2,571	130	77	210	25	3,593	168	97	29	3
Deaths	1	—	2	1	—	—	—	1	—	—
Ophthalmia neonatorum	56	—	13	—	—	82	3	22	—	—
Deaths										
Paratyphoid fever	8	—	9 (B)	—	—	12	—	1	—	—
Deaths	—	—	—	—	—	—	—	—	—	—
Pneumonia, influenzal†	539	45	11	2	1	502	19	2	—	6
Deaths (from influenza)	10	1	1	—	—	9	1	5	—	—
Pneumonia, primary			179	15				195	23	
Deaths		35		8	7		15		8	2
Polio-encephalitis, acute	1	—				2	1			
Deaths		—					—			
Poliomyelitis, acute	7	—	5	1	—	6	3	2	—	—
Deaths		—					—			
Puerperal fever		—	16		—		4	24		—
Deaths										
Puerperal pyrexia‡	164	11	14	—	1	125	8	18	—	1
Deaths		1					—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,390	87	164	19	52	1,959	132	209	40	4
Deaths	—	—	—	—	—	—	—	—	1	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	3	—	4	5	2	8	2	4	12	6
Deaths	2	—	—	—	—	1	1	—	—	—
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough*	2,325	171	62	73	15	2,284	100	94	28	3
Deaths	16	3	5	1	2	8	2	3	1	—
Deaths (0–1 year)	312	48	63	31	16	293	43	42	30	2
Infant mortality rate (per 1,000 live births)										
Deaths (excluding still-births)	4,717	1,217	585	196	142	3,591	560	601	194	11
Annual death rate (per 1,000 persons living)			13·4	12·7	§			13·5	12·8	§
Live births	7,579	914	935	411	285	6,418	762	995	386	23
Annual rate per 1,000 persons living			19·0	—	§			20·3	25·4	§
Stillbirths	194	20	36			172	20	28		
Rate per 1,000 total births (including stillborn)			37					28		

* Measles and whooping-cough are not notifiable diseases in Scotland, and returns are therefore an approximation only.

† Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

‡ Includes puerperal fever for England and Wales and Eire.

§ Owing to evacuation schemes and other movements of population, birth and death rates for Northern Ireland are no longer available.

Letters, Notes, and Answers

All communications with regard to editorial business should be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 2111. TELEGRAMS: *Aitiology Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

Authors desiring REPRINTS should communicate with the Publishing Manager, B.M.A. House, Tavistock Square, W.C.1, on receipt of proofs. Authors over-seas should indicate on MSS. if reprints are required, as proofs are not sent abroad.

ADVERTISEMENTS should be addressed to the Advertisement Manager, B.M.A. House, Tavistock Square, London, W.C.1 (hours 9 a.m. to 5 p.m.). TELEPHONE: EUSTON 2111. TELEGRAMS: *Articulate Westcent, London.*

MEMBERS' SUBSCRIPTIONS should be sent to the SECRETARY of the Association. TELEPHONE: EUSTON 2111. TELEGRAMS: *Medisecra Westcent, London.*

B.M.A. SCOTTISH OFFICE: 7, Drumsheugh Gardens, Edinburgh.

ANY QUESTIONS?

Haemorrhage after Gastro-jejunostomy

Q.—A man now aged 58 had, at the age of 30, a gastro-enterostomy for duodenal ulcer. He kept in good health until the age of 52, when he had recurrence of dyspepsia. Five months ago he had a severe haemorrhage, mostly in the form of melaena, but also with some vomiting of dark blood. I should be glad of answers to the following questions in relation to this case: (1) Is there any means of telling whether the haemorrhage was from the original ulcer or from a new one on the stoma (peptic) or a new one in the duodenum? (2) Does it make any difference to the treatment where the site of the ulcer is, and, if so, what? (3) Have there been any recent advances in treatment beyond the usual dieting, etc., and the taking of magnesium trisilicate between meals? (4) Is the taking of vitamin A really helpful in healing the ulcer, and is the taking of vitamin C in tablet form (ascorbic acid) undesirable?

A.—Haemorrhage with or without indigestion after gastro-jejunostomy for duodenal ulcer almost always comes from the anastomotic margin and not from the duodenum. In the absence of indigestion it may be from acute ulcers associated with marginal gastro-jejunitis; when indigestion is present, it is likely to be from a chronic gastro-jejunal or jejunal ulcer. The treatment is similar to that of duodenal ulcer, but rest in bed with hourly feeds, with or without magnesium trisilicate or aludrox and atropine, must generally be continued for an even longer period, generally 6 to 8 weeks instead of 4 to 6 weeks. After this the patient must remain permanently on a suitable post-ulcer regime, the most important feature of which is the taking of food at intervals of not more than 2 hours throughout the day. Additional vitamins do not hasten recovery, but when fresh orange or tomato juice is not available 50 mg. ascorbic acid dissolved in milk should be given on account of the deficiency of the diet in vitamin C.

Tenderness after Shaving

Q.—In these days of make do and try-to-sharpen safety razor blades, what is the most effective treatment of the facial skin (especially of that of the tender upper lip) before and/or after shaving?

A.—A brushless shaving cream may be substituted for soap, or an electric razor used if obtainable. Zinc cream as a basis for calamine lotion, used after shaving, will probably be found curative.

Liver Stores and Ketosis

Q.—Is there any evidence that, in the absence of starvation, emotion or exertion or excitement can deplete the liver of glycogen so completely that there is not much left for conversion into blood sugar? A short biochemical explanation of ketosis, which is fairly common in children, would be of interest.

A.—The glycogen content of the liver depends upon input and output balance, and upon nervous and endocrine controlling factors. It is undoubted that sympathetic activity associated with emotion, excitement, or exertion may cause a temporary decrease in the glycogen stores. If, however, adequate food intake is maintained (which is not always the case under these conditions) it is improbable that the sympathetic activity could be sufficiently prolonged to maintain a state of glycogen depletion. The effect of emotion, excitement, or exertion on the complex interrelationships between insulin and the pituitary and adrenal cortical hormones is not known. The generally accepted view of ketosis is that the major source of ketonic bodies is fat. If the proportion of carbohydrate to fat being burnt is lowered, there appears to be difficulty in the combustion of aceto-acetic acid. The accumulating aceto-acetic acid is detoxicated by reductases to form beta-hydroxy-butyric acid and by decarboxylases to form acetone.

Imbalance between carbohydrate and fat may occur due to exogenous factors, such as a high fat diet, or to endogenous causes, such as depletion of carbohydrate stores in starvation, lack of insulin with consequent interference with carbohydrate oxidation, or to excessive mobilization of fat as by ketogenic hormones. Ketones may also arise from other sources than fat, such as the non-nitrogenous residue of aromatic amino-acids. In ordinary cases of ketosis found in clinical practice, however, it is probable that an upset of fat and carbohydrate metabolism is the main underlying cause.

Hallucinations and Heart Failure

Q.—In two cases of decompensated heart failure, hallucinations of a vivid and realistic character developed, and not many days after their first appearance both patients died. How is the occurrence of these hallucinations to be explained, and are they of any prognostic significance?

A.—Hallucinations of all kinds are liable to appear when the functions of the cortex are interfered with. Deficient oxygenation of the blood is one of the commonest causes of such an interference, and seems to be particularly common in the causation of visual hallucinations. For instance, many perfectly healthy people have vivid visual hallucinations, of the unreality of which they are aware, in the half-dreamy state before going off to sleep (hypnagogic hallucinations). Some people can even entertain themselves with a sort of automatic cinema-show before dropping off. In the patients referred to, the appearance of the hallucinations was the sign of an increase in cardiovascular inefficiency, and so could be legitimately regarded as of bad prognostic significance. In these patients there may have been also other signs of impaired cortical function—e.g., some failure of memory for recent events or of the power of concentration.

Treatment of Anxiety Symptoms

Q.—A man of 31, suffering from anxiety symptoms consisting mainly of excessive nervousness and tremulousness in company, apparently the result of unknown factors originating in early childhood, has been advised to undergo a course of electric shock treatment. Present circumstances make it impossible for him to undergo a prolonged analysis, and it is suggested that shock treatment, though not a cure, may somewhat alleviate the symptoms. He is an intelligent type, whose interests are mainly on mathematical and other intellectual lines, and his fear is that the treatment may impair his faculties. Though not entirely disabling, his condition is distressing because it cuts him off from social contacts and is a serious handicap in his career, and he is therefore quite willing to face the risk of fractures, which have been mentioned as a possibility. What advice can be given as regards risks, immediate and late? Presumably shock therapy would not interfere with analytical or other treatment at a later date.

A.—Electrical convulsion therapy is not desirable. The evidence that it helps this class of condition is practically *nil*, and its administration here would be the purest empiricism. The possibility of such treatment injuring intellectual powers must always be borne in mind. Psychotherapy of an intensive sort is the correct treatment, and it may not prove to be a prolonged matter.

Galactorrhoea

Q.—Could you suggest treatment for a serous discharge from both nipples of a patient who has been so afflicted for eight years, since an unsuccessful pregnancy (premature stillbirth)? Hormone therapy has been unsuccessful. At no time has there been blood. There is no apparent mastitis.

A.—The history suggests that the discharge is due to a persisting secretory function of the mammary glands and that it is either milk or analogous to it. Galactorrhoea, as this symptom is called, is rare, but extraordinary examples have been recorded from time to time through the centuries, sometimes even in virgins and men. It can result from lesions in the pituitary gland or the adjacent parts of the brain, and in particular is associated with acromegaly. Ovarian tumours such as lutein cysts have also been reported as causing it. It sometimes follows ovarian failure and has been described as a cyclical phenomenon coinciding with, or replacing, menstruation. The above possibilities should be excluded, but a more simple explanation should be kept in mind. Lactation can be maintained almost indefinitely by local stimulation of the breasts, and if the patient is continually attempting to express the discharge to confirm its presence, that might be enough to account for its persistence.

If a cause cannot be found, treatment is difficult. The breasts should be well supported by a brassière and all forms of manipulation avoided. Assuming that it is oestrogenic hormone therapy which has failed, it would be worth while trying the effect of androgens, either testosterone propionate intramuscularly or methyl testosterone sublingual tablets. The dose will be a matter for trial, but large amounts may be required. Alternatively, ethisterone might be helpful. Iodine and iodides have also been used, apparently with good effect in some cases.

Aniline Poisoning

Q.—*(1) Is it known what abnormal compound of haemoglobin exists in the blood in cases of anilism in those cases of poisoning by aniline or its compounds, and in the particular case of T.N.T.? (2) Is this compound formed by direct combination in the alveoli of the lungs when fine dust or fumes of T.N.T. are inhaled, or is it formed by the action on the blood of T.N.T. absorbed through the skin, or by both channels? (3) Is the bone marrow affected in these cases of anilism, and, if not, why does cyanosis persist for several weeks when cases are removed from contact. (4) Can the abnormal haemoglobin compounds carry any oxygen whatever? (5) Does toxic hepatitis play any part in the production of cyanosis in cases of anilism?*

A.—(1) The compound formed is chiefly methaemoglobin, with possibly some nitrohaemoglobin. (2) These compounds are formed by the action of T.N.T. on the blood after absorption through both skin and lungs, and possibly to some extent by ingestion. (3) In cases of pure anilism, without the rarer incidence of true aplastic anaemia, there is usually a depression of red-cell elements, with a consequent anaemia, accompanied by a fall of haemoglobin. True aplastic anaemia may be a feature of T.N.T. poisoning without cyanosis. (4) Methaemoglobin and nitrohaemoglobin carry no oxygen, but it must be remembered that only a certain percentage of haemoglobin is converted into these abnormal compounds. (5) Toxic hepatitis has no direct connexion with cyanosis, but the liver, the bone marrow, and the haemoglobin may be attacked simultaneously as well as separately.

Tap Water and Blood Transfusion

Q.—*A blood transfusion was done on a patient. Tap water was used for rinsing and boiling rubber tubing, needle, and glass tube. The patient had a severe reaction during the transfusion and was quite ill for 48 hours. The transfusion was of fresh blood and was cross-matched. Was his reaction due to the tap water used? Is freshly distilled water now always used, even at the front? Are many people so allergic to tap water? Do these reactions occur even in glucose-saline transfusions if tap water is used?*

A.—It is maintained by certain workers that London tap water can be used for rinsing and cleaning transfusion apparatus. The Emergency Transfusion Service would not use the tap water for rinsing and boiling rubber tubing, needle, and glass tube unless the apparatus was subsequently dried and autoclaved. It is possible, of course, that a small amount of dried protein from previous transfusions remained in the apparatus, but the most likely explanation of the reaction is the use of tap water. At the front dry sterilized apparatus is provided. I should expect reactions to occur in glucose-saline transfusions if tap water were used, but know of no evidence on this point.

Sunlight and Tuberculosis

Q.—*Is sunlight, artificial or natural, harmful in cases of active pulmonary tuberculosis? Can it be given with safety in gradually increasing doses? Is it the case that those people who tan as the result of exposure to the sun will not derive any harm from this practice, and vice versa? What is the position with regard to tuberculosis of other regions?*

A.—The use of sunlight, artificial or natural, is dangerous in active pulmonary tuberculosis. Its value is rather problematical and is outweighed by the danger unless prescribed by an expert. It is not the case that those people who tan easily as a result of exposure to the sun will derive no harm from this practice, though they are less likely to do so. The use of carefully graduated doses of sunlight in cases of bone-and-joint, glandular, and peritoneal tuberculosis is very valuable, but the best results will be obtained by those with experience. The questioner is recommended to consult *Actino-Therapy* by Rollier for details.

Cysts of Buccal Mucous Glands

Q.—*For the past nine months a patient has had recurrent cysts of the buccal mucous glands. The condition was limited to the inside of the lower lip to the left of the midline—approximately over the distribution of the terminal branches of the left mandibular nerve. Recently the opposite side has been affected also. The cysts periodically break, liberating a clear fluid which shows nothing microscopically. Many local treatments and attention to general health have been tried with no improvement. Occasionally only, say, six cysts are present, sometimes many more.*

A.—Cysts of the muciparous glands of the lips are not uncommon. They are retention cysts, and when recurrence occurs, as in the present case, trauma, lip-biting, or incomplete removal should be suspected. The treatment is surgical. Single cysts can be shelled out individually, but when there is a general cystic condition of the glands a flap should be turned back and the mass dissected out.

INCOME TAX

Assistant

"X. Y. X." employed an indoor assistant at £450 a year, but he has recently become an outdoor assistant receiving an additional £104, making £554 a year. (*a*) If he is paid the £104 separately to meet the rent he will have to pay will the £104 be exempt? (*b*) His new house will be used for professional purposes; what expenses can the assistant claim for this?

*** (*a*) No; the £104 is assessable to income tax and is liable to deduction of tax under "pay as you earn." (*b*) Assuming that the assistant is required by the conditions of his employment to use his house for professional purposes, he can claim to deduct a reasonable proportion of the general cost of the house, rent, rates, lighting, etc. What a reasonable proportion would be must of course depend on the particular facts.

LETTERS, NOTES, ETC.

Sterilization of Syringes

Dr. E. H. ROBERTS (Hythe, Southampton) writes: I have been in the habit of keeping my syringe in its container in 50% "dettol." I change the needle when it is blunt and the dettol every fortnight. For some years I did not disinfect the skin (I do now with the dettol in the container for the patients' benefit and to be on the safe side). I carry out this method for all kinds of injections—mainly T.A.B., T.A.F., A.P.T., whooping-cough vaccine, etc. If I do what I call a "dirty" injection, I then change the needle and soak the syringe in pure dettol for half an hour. After using my syringe for a fortnight in this way I had the dettol in the container and an injection of sterile saline, which I sucked up in the syringe, cultured (2 of each). These showed growth of *B. subtilis*, no other organisms being found. *B. subtilis*, I understand, is regarded as harmless to man on injection. I have used this method for ten years for several thousand injections with, so far, no ill effect. It is labour-saving and, I hope, is fool-proof.

Offers of Hospitality for Doctors' Children

The following additional offers have been received from doctors willing to accommodate the children of colleagues practising in dangerous areas. Dr. A., who practises in a village in Lincolnshire, could take two children, preferably girls, for the period of the school holidays, or longer if necessary. He has two daughters aged 15 and 9, who will be returning to school towards the end of September. Alternatively, Dr. A. offers to accommodate a doctor and his wife, or to exchange practices for a fortnight with a doctor in the London area. Dr. B., of Birmingham, offers accommodation for two boys, preferably Scottish. Dr. C., who is in practice near Plymouth, has a daughter of 10 and offers to accommodate a girl between the ages of 9 and 11 for the holiday period. Dr. D., of Sheffield, who has a young daughter, would be glad to accommodate two children, preferably over 10 years of age. Dr. E., practising in Cornwall, offers to take a child over 7, either a boy or a girl; he has three children of his own. Any readers interested in these offers may communicate with the Secretary at B.M.A. House, Tavistock Square, W.C.1.

The Flying Bomb: Exchange of Duties

A doctor living in the North in "country surroundings that are particularly attractive just now" writes to suggest that "we doctors who live in safe areas should be allowed to take our turn at the casualty services and give our overstrained colleagues in London a much-needed rest by changing places for a few weeks." He asks if it could be arranged for him to do his share during the present flying bomb attacks. He has already been put in touch with a London colleague, but we publish his suggestion as there may be others who would like to follow his example.

Baby wanted for Adoption

Dr. E. W. DEWEY (214, London Road, Portsmouth) writes: I wonder if any of your readers know of a baby requiring adoption. The child should be a boy, between 2 and 6 months, healthy, and of good stock, and preferably resident somewhere in the South of England. I should be glad if anybody knowing of such a child would communicate with me.

Correction

Dr. ARTHUR F. COCA (New York), whose letter on vitamin B deficiency in allergic patients appeared last week (p. 128), asks that the following should be substituted for the last sentence of paragraph 2—that is, after the word "prescribed": "The daily dose required for complete relief of the symptoms of B deficiency is from four to seven cakes of Fleischmann's standard yeast." In a covering letter Dr. Coca comments that "the small dose of one and one-half cakes of yeast was satisfactory only with a supplemental small dose of liver extract."